A Dissertation on

CLINICAL PATTERN OF DIABETIC FOOT INFECTIONS AND THEIR MANAGEMENT

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr.T.MOHANA PRIYA** on " **CLINICAL PATTERN OF DIABETIC FOOT INFECTIONS AND THEIR MANAGEMENT"** during her M.S.(General Surgery) course from May 2005 to March 2008 at the Government Kilpauk Medical College and Government Royapettah Hospital, Chennai.

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CONTENTS

1. INTRODUCTION

1.0 Diabetes mellitus

In the years between 1958 and 1993, the number of people diagnosed with diabetes multiplied five – fold[1]. In 1994, 135 million patients world - wide were living with Diabetes mellitus. By the year 2025, it is estimated that this figure would increase to more than 300 million[2].

1.1 Diabetes Mellitus - Definition

Diabetes mellitus is a chronic metabolic disorder, predominantly of carbohydrates, which has hereditary and environmental risk factors. According to the criteria of WHO and the ADA (American Diabetes Association) of 1997[3], a diagnosis can be established on the basis of fasting plasma glucose levels of :

1. 7.8 mmol/L (126 mg/dl) or above (with or without the presence of the classic signs such as polydipsia, tiredness, unexplained weight loss or pruritus).

2. 11.1 mmol/L (200mg/dl) and above measured at random and coexisting with the disease symptoms mentioned previously.

3. 11.1 mmol/L (200mg / dl) measured two hours after a standardized oral glucose tolerance test.

2.0 AIM AND OBJECTIVES OF THE STUDY

With the projected 14% prevalence rate of Diabetes Mellitus in Indian population and about 5 – 10% of them developing foot infections and associated foot lesions, it becomes imminent for the health care system to put in practice a logistically feasible management strategy for Diabetic foot in Government Hospitals. This becomes important in view of the fact that over 80% of general population in India approach only Government Hospitals for their health care requirements.

With this aim in mind, the present study was planned and conducted. The objectives of the project are:

2.1. To study the clinical pattern of foot infections in Diabetic patients.

2.2. To study the effect of Glycaemic status in controlling infection

2.3. To analyze the risk factors leading to complications in Diabetic Foot-infections

2.4. To study the outcome of the treatment modalities and suggest a patient friendly hospital management strategy for Diabetic foot

3.0 REVIEW OF LITERATURE

3.1 Definition and History of the Diabetic foot

3.1.1 Definition

The World Health organization defines the diabetic foot as an infection, ulceration and / or destruction of deep tissues associated with neurological abnormalities and various degrees of peripheral vascular disease in the lower limb. In Dutch consensus, the diabetic foot is defined as a diversity of foot abnormalities caused by neuropathy, micro- angiopathy, limited joint mobility and other consequences of metabolic disturbances, mostly occurring in combination, in patients with diabetes mellitus. Both definitions are descriptions of causal factors and resulting foot disorders. This emphasizes that the diabetic foot is more a syndrome rather than a diagnosis.

3.1.2 History

`Pyrce described, `a case of a perforating ulcer in diabetes and atactic symptoms, as early as 1887[4]. In 1934, Elliot Joslin, one of the pioneers of diabetology, published an article entitled, `The menace of diabetic gangrene', in which, Joslin described the common causes of diabetic foot lesions[5], and he wrote, "that gangrene is not heaven sent, but is earth born". However, it was not until the 1950s that diabetic neuropathy, ischemia and infection were finally recognized as precondition of foot complications in diabetics - facts that still hold good today.

3.2 Epidemiology of the diabetic foot syndrome

A quarter of the diabetic population is at increased risk of foot injuries as a result of the presence of diabetic neuropathy or an arterial circulatory disorder. Every year 3 to 7% of diabetics suffer a foot lesion for the first time.

Foot ulcers occur in approximately 15% of people with diabetes which accounts for 25% of all hospital admissions with the hospital stay being 60% longer than the stay for other causes and the risk of amputation is 15 to 40 times greater in diabetics than in others[6].

Diabetic foot ulcers account for more than 50% of non traumatic amputations and are associated with high rates of mortality, re-amputation and contra lateral limb amputation.

India has 30 million diabetics at present and in the year 2025 India is predicted to have 57 million diabetics.

3.2.1 Incidence in India

Foot Ulcer	:	1-4%
Toe amputation	:	2.6%
Below knee amputation	:	1.6%
Prevalence of diabetic foot in India	:	5.3 - 10.5%

3.2.2 Socio - economic impact of the diabetic foot syndrome

Overall, the costs generated by diabetes are about three times as high as those produced by non - diabetics. Foot complications constitute a major proportion of these.

With primary healing, about 30% of the total cost derives from hospitalization, but where amputation is required this figure is 65% to 80%. The average healing duration for diabetic foot lesions is about four months.

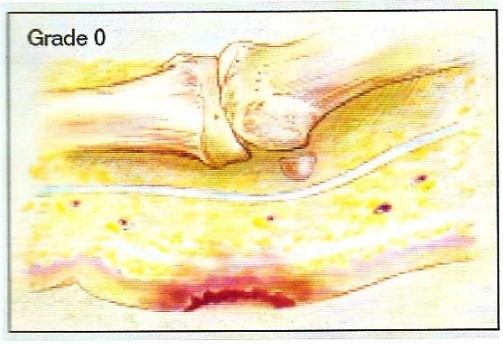
Ten percent of all lesions persist for more than one year, which incurs further costs for outpatient care. Fifteen percent of all foot ulcers in diabetics do not heal before the patient's death[7].

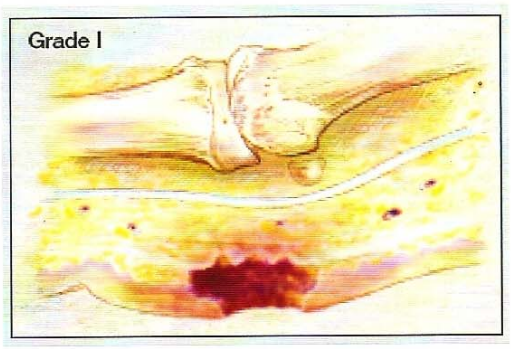
Most important cost item is namely the "cost" to the patient themselves in terms of the emotional trauma suffered and the loss of quality of life and independence[8a].

3.3 CLASSIFICATION OF DIABETIC FOOT.

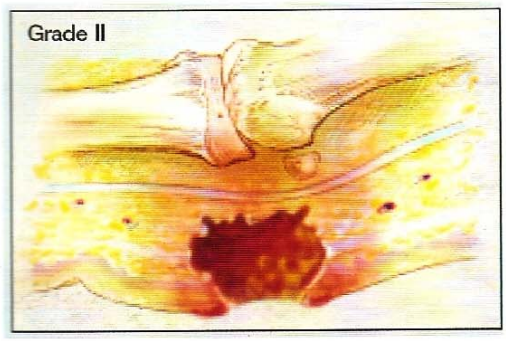
3.3.1 The Meggitt – Wagner classification is the most well-known and validated system for foot – ulcers[8b].

Grade	Description
Grade 0	Pre or post-ulcerative lesion completely epithelialized.
Grade 1	Superficial, full thickness ulcer limited to the dermis, not extending to the subcutis
Grade 2	Ulcer of the skin extending through the subcutis with exposed tendon or bone and without osteomyelitis or abscess formation.
Grade 3	Deep ulcers with osteomyelitis or abscess formation.
Grade 4	Localized gangrene of the toes or the forefoot.
Grade 5	Foot with extensive gangrene





WAGNER GRADING OF DIABETIC FOOT LESIONS



3.3.2 The University of Texas classification system[9] for Diabetic foot wounds

Grade

Stage	0	1	2	3
A	Pre or post-ulcerative lesion completely epitheliasied	Superficial Wound not involving tendon, capsule or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	with infection	with infection	with infection	with infection
C	with ischemia	with ischemia	with ischemia	with ischemia
D	with infection & ischemia	with infection & ischemia	with infection & ischemia	with infection & ischemia

3.3.3 Edmonds & Foster classification

Based on clinical tests and determination of the ankle brachial pressure index.

Foot ulcers → Neuropathic
Foot ulcers → Neuro-ischemic

3.3.4 **Broadsky** suggested the **`depth-ischemia classification'**

Which is a modification of the Meggitt - Wagner Classification.

A - Not ischemic

B - Ischemic without gangrene

C - Partial gangrene of the foot

D - Complete foot gangrene with grades 1-3 (similar to the Meggitt – Wagner classification)

3.3.5 Macfarlane and Jeffcoate proposed the **S(AD)AD classification** for diabetic foot ulcers[10].

According this system, ulcers are classified on the basis of

Size, (**A**rea & **D**epth)

Presence of **S**epsis

Arteriopathy

and

Denervation

3.4 RISK FACTORS FOR FOOT COMPLICATIONS IN DIABETICS:

Classification of categories of diabetic patients based on the risk for ulceration [11].

3.4.1 Risk category

0. Protective sensation is intact, the patient may have foot deformity.
1. Loss of protective sensation.
2. Loss of protective sensation high. Plantar pressure or callosities or history of foot ulcer.
3. Loss of protective sensation and history of ulcer and severe foot or toe deformity and / or limited joint mobility; significant peripheral vascular disease.

3.5 CLINICAL PATTERN OF DIABETIC FOOT LESIONS

(I) Infections

(II) Ulcers

(III) Gangrene

(IV) Joint lesions

3.5.1 Infections

Diabetic foot infections are categorized as mild, moderate or severe.

3.5.1.1 Mild infections are superficial infections confined to the skin and the subcutaneous fat with minimal or no purulence or cellulitis. This is otherwise known as **non-limb threatening infections**

3.5.1.2 Moderate infections are deep and may involve fascial, muscles, tendons, joints or bones. They may present as cellulitis of 0-2 cms in diameter or a plantar abscess, and they may cause systemic symptoms. They impose a certain risk of amputation.

3.5.1.3 Severe infection of a foot ulcer is a deep infection with more than 2 cm of cellulitis, lymphangitis, gangrene and or necrotizing fascitis, threatening limb loss and causing systemic toxicity. Absence of symptoms or signs of systemic illness does not exclude a limb threatening infection.

Moderate and Severe infections are collectively known as **limb-threatening infections.**

3.5.1.4Bacterial Infections

Staphylococcus aureus or β - hemolytic streptococci, pathogens that colonize the skin of diabetic patients are the causative agents of acute infections in antibiotic-naive patients, and are nearly always the cause of cellulitis in non-ulcerated skin.

Staphylococcus aureus is the most commonly recovered pathogen in most infections in which a single agent is isolated. Polymicrobial cultures, with an average of five or six organisms are often obtained from patients with chronic lesions especially when they have been treated with antibiotics for sometime, anerobes, mostly Bacteroides Sp. and various anaerobic gram-positive cocci are often isolated from deep necroses.

Proteus Spp. and Escherichia coli predominate among gram-negative bacilli and Pseudomonas is often isolated from indurated, wet wounds.

In severe infections, Gram-negative pathogens and anaerobes predominate versus gram positive pathogens and Enterobacteriaceae, which are usually isolated from mild infections. Severity of infection does not predict the causative organism.

Non limb threatening infections are usually caused by gram positive cocci, typically Staphylococcus aureus and Streptococcus spp. In hospitalized patients with diabetic foot infections, methicillin–resistant Staphylococcus aureus as well as Enterococci are more prevalent.

3.5.1.5NECROTIZING FASCITIS: is a progressive rapidly spreading infection located in the superficial and deep fascial planes with secondary

necrosis of the subcutaneous tissues. Due to the presence of gas forming organisms, subcutaneous air is often noticed in X-ray. The speed of spread is directly proportional to the thickness of the subcutaneous layer. Necrotizing fascitis moves along the deep fascial plane, therefore requiring rapid treatments.

3.5.1.6Fungal Infections

Fungal infections develop as a result of poor foot hygiene, hyperhydrosis and accumulation of moist debris in the webs. Interdigital Tinea pedis is the most common form of chronic fungal foot infection. Itching, Redness, scaling, erosion and soaking of the skin with fluid usually occur, while in the late phase the redness subsides. Trichophyton metagrophytes, Trichophyton rubrum or Epidermophyton floccosum may be found.

3.5.1.7 BONE INFECTIONS – OSTEOMYELITIS

All patients with deep or long-standing ulcers should be evaluated for osteomyelitis. The possibility of an ulcer being complicated by osteomyelitis increases when the diameter of the ulcer exceeds 2cms and the depth in greater than 3mm, the possibility of complications becomes even higher when the white blood cell count, the erythrocyte sedimentation rate and the c-reactive protein levels are high. When a chronic recurrent perforating ulcer is neglected for a longtime, the bacteria invade the base of the lesion, then the fascial plane, later the periosteum leading to osteomyelitis[13].

3.5.2 ULCERS

Ulcers – Another pattern of clinical presentation in Diabetic foot lesions and it is often associated with infection.

Diabetic Ulcers are classified as

a. Neuropathic ulcers

b. Ischemic ulcers

c. Neuro ischemic ulcers

3.5.2.1NEUROPATHIC ULCERS

- Develop at areas of high plantar pressures. (Metatarsal heads, plantar aspect of the great toe, heel or over bony prominences in a charcot – type foot). Neuropathy is present in about 85-90% of foot ulcers in diabetic patients.

- Are painless, unless they are complicated by infection.

- There is callus formation at the borders of the ulcer. Its base is red, with a healthy granular appearance.

- On examination evidence of peripheral neuropathy (hypoesthesia or complete loss of sensation of light touch, pain, temperature and vibration, absence of Achilles tendon reflexes, abnormal vibration perception threshold, often above 25v, atrophy of the small muscles of the feet, dry skin and distended dorsal foot veins) is present. However

the pattern of sensory loss may vary considerably from patient to patient.

- The foot has normal temperature or may be warm. Peripheral pulses are present and the ankle brachial pressure index is (N) or above 1.3.

3.5.2.2ISCHEMIC ULCERS:

- Ischemia is a major factor in 38-52% of cases of foot ulcers. These ulcers develop on the borders or the dorsal aspect of the feet and toes or between toes. They are usually painful.

- There is usually redness at the borders of the ulcer. Its base is yellowish or necrotic (black). There is history of intermittent claudication.

- On examination signs of peripheral vascular disease (skin is cool, pale or cyanosed, shiny and thin, with loss of hair and onychodystrophy; peripheral pulses are absent or weak, the ankle – brachial index is <0.9) are present.

- Non-invasive vascular testing (duplex or triplex ultrasound examination, segmental pressures measurement, plethysmography) and angiography confirm peripheral vasculopathy.

3.5.2.3Neuro-Ischemic Ulcers (Mixed Etiology ulcers)

- Neuro ischemic ulcers have a mixed etiology ie., neuropathy and ischemia.

3.5.3 GANGRENE

Gangrene implies death with putrefaction of macroscopic portions of the tissue.

3.5.3.1Gangrene of Toes

This is by far the most commonly noted type of lesion often starts from an unnoticed minor injury. It is usually of the wet type when there is infection but dry type of gangrene is also seen especially when there is associated vascular disease.

3.5.3.2Gangrenous Patches

These occur in the pressure areas of the foot, most commonly over the heel, the 1st metatarsal medially and the base of the 5th metatarsal laterally. Small areas of gangrene are also seen in non pressure areas due to atheromatous debris. They are also seen in the interdigital clefts which are often missed during a routine examination.

3.5.3.3Diabetic Gangrene

This is a term specifically given to a gangrene of a fully vascularized foot. It is usually rapid on onset, painless with large areas of necrosis.There may be associated systemic illnesses, signs of deep infection are present but the striking feature is that the ankle pulses will be well felt.

3.5.4 JOINT LESIONS

Charcot Osteoarthropathy (Neuro-Osteoarthropathy, Charcot arthropathy, Diabetic Neuropathic osteoarthropathy) (DNOAP)

This represents one of the most serious complications of diabetes. It's prevalence is between 1 & 7.5%, bilateral involvement has been reported to occur in 6-40% of patients in several series[15]. The development of this complication depends on peripheral somatic and autonomic neuropathy, together with adequate blood supply to the foot. Mean age of presentation is approximately 60 years and the majority of the patients have diabetes of more than 15 years duration. Men & women are affected equally.

3.6 PATHOPHYSIOLOGY OF DIABETIC FOOT LESIONS

Ischemia, neuropathy, infection and sustained hyperglycemia are the principal pathogenic factors.

3.6.1 Role of Vasculopathy

Atherosclerosis tends to occur with greater frequency and severity and appears earlier in diabetics than in age - matched controls.

Diabetics characteristically have two different types of arterial changes : Large vessel (macroangiopathy) and small vessel (microangiopathy). There are qualitative differences in mucopolysaccharides, calcium and cholesterol compared with non - diabetics. The macrovascular lesion in "Garden - Variety" atherosclerosis. The disease is much more extensive and more commonly associated with medial calcific sclerosis in diabetics than in non – diabetics[16].

Diabetic microangiopathy involves arteries smaller than 115 micrometer in diameter. The severity and extent of the small vessel lesion distinguish diabetics from non diabetics. The hall mark of diabetic microangiopathy is PAS positive thickening of the capillary wall.

DIABETIC PERIPHERAL VASCULAR DISEASE HAS PREDILECTION FOR TIBIO- PERONEAL VESSELS. ALL THE TIBIAL ARTERIES ARE OCCLUDED WITH SPARING OF THE DORSAL PEDAL OR COMMON PLANTAR ARTERY[17].

3.6.2 ROLE OF NEUROPATHY

Neuropathy is an important factor in the development of diabetic foot problems. Peripheral neuropathy may well be related to the quality of glycemic control. Hyaline arteriosclerosis has been found in intraneural arterioles in diabetics.

Of the various manifestations of diabetic neuropathy, three are of relevance to the lower limb (i) the acute sensory; (ii) chronic sensorimotor and autonomic neuropathies. A progressive, stock and glove symmetrical loss of vibration, temperature and pain perception is typical of the chronic sensorimotor form of the disease[18].

The neuropathic diabetic foot is at greater risk as there is no protective sensation, minor trauma is unnoticed until there is significant ulceration, infection or bone injury. And also because of intrinsic foot muscle atrophy and secondary foot deformities, there is an alteration in the weight distribution and biomechanics of foot function that leads to pressure points, callus formation and skin breakdown.

3.6.3 Other Risk factors

Other pathogenic factors include hyperlipidemia, hypertension, smoking, the secondary consequences of hyperglycemia, obesity, genetic factors and hypercoagulability[19]. Diabetic foot complications depends on both the duration and severity of hyperglycemia over years. Foot deformities such as pesplanus, pescavus, bunionette, clawtoe, over riding toe deformity, mallet toe are more common in diabetics due to muscle atrophy and limited joint mobility[20,21].

Apart from the above obvious clinical predisposing risk factors, recent studies have revealed that very complex mechanisms are involved at the tissue - molecular level, which prevent normal healing processes. Many chemo - cytokines are involved, including matrix metalloproteinases, serine proteinases, integrins, chemokines, replicative cell senescence, growth factors and adult stem cells[22].

Diabetic patients with tissue injury initially display impairment in the immune system response with reduced chemotactic effects to recruit inflammatory cells into the damaged tissues, thus, slowing down healing and increasing the risk of bacterial infection. Following this initial period, when the inflammatory response is eventually established, the process switches to an exacerbation of inflammation and proteolysis. The result of prolonged exposure to hyperglycemia also generates glycation of proteins and disturbances of cell responses, thus, further hindering the process of fibrosis and tissue repair[23]. Recent molecular studies on chronic diabetic ulcers indicated that more specific processes may be involved. For example, it has been found that leucocytes are prevented from ready entry and accumulation in the ulcers, which therefore, fail to achieve normal healing[24].Other studies on the specific properties of fibroblasts from patients with chronic diabetic ulcers showed that these cells

were different from those taken from patients without chronic ulcers in that the high molecular weight hyaluronic acid in the pericellular matrix was much more concentrated. The unique property of the fibroblasts might predispose these patients to chronic ulcer formation[25].

Clinical Pathways leadings to foot ulceration[26].

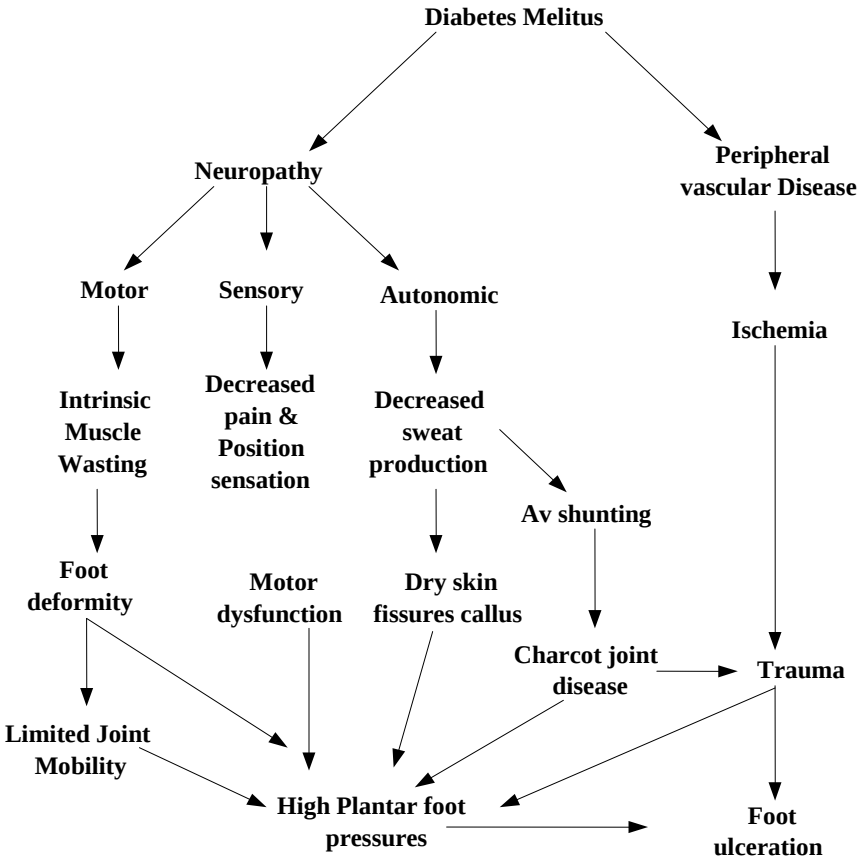


Table. 1.Risk factors for foot ulceration and infection

Risk factor	Mechanism of Injury or Impairment
Peripheral motor neuropathy	Abnormal foot anatomy and biomechanics, with clawing of toes, high arch and subluxed metatarsophalangeal joints, leading to excess pressure, callus formation and ulcers.
Peripheral sensory neuropathy	Lack of protective sensation, leading to unattended minor injuries caused by excess pressure or mechanical or thermal injury.
Peripheral autonomic neuropathy	Deficient sweating leading to dry, cracking skin.
Neuro-osteoarthropathic deformities (ie, charcot disease) or limited joint mobility	Abnormal anatomy and biomechanics, leading to excess pressure, especially in the midplantar area.
Vascular insufficiency	Impaired tissue viability, wound healing and delivery of neutrophils.
Hyperglycemia and other metabolic derangements	Impaired immunological (especially neutrophil) function and wound healing and excess collagen cross - linking.
Patient disabilities	Reduced vision, limited mobility and previous amputation(s).
Maladaptive patient behaviours	Inadequate adherence to precautionary measures and foot inspection and hygiene procedures, poor compliance with medical care, inappropriate activities, excessive, weight bearing and poor foot wear.
Health care system failures	Inadequate patient education and monitoring of glycemic control and foot care.

3.7 EVALUATION OF DIABETIC FOOT LESIONS:

3.7.1 Evaluation of Neuropathy:

Loss of protective sensations is almost always found in diabetic ulceration. Neuropathy may be detected by following methods[27] :

* Vibration sensitivity : Tested by Biothesiometer and Tuning fork test

* Temperature discrimination : tested by Tip Therm.

Biothesiometer (Vibration Perception threshold meter)

This is a hand - held probe whose tip vibrates at 100 HZ. The voltage supplied to the probe can be adjusted from 0 to 50V. The probe is placed against the skin and voltage increased till the patient perceives the vibration. Mean of three readings is used to determine the vibration perception threshold for each foot. Normal readings should be less than or equal to 25v.

Semmes - Weinstein monofilament : is a valuable, easy to use tool. The monofilament is a long nylon wire, the tip of which is pressed against the skin to the point of buckling for at least one second. The points of testing are plantar aspects of 1st, 3rd and 5th digits, the plantar aspects of 1st, 3rd and 5th metatarsal heads, plantar midfoot medially and dorsally and the plantar heel (10 sites totally). Neuropathy is said to exist when 4 out of there 10 sites show absence of sensation when the wire is pressed against the skin. Temperature discrimination can be tested economically and reproducibly with the tip therm. With a combination of the tuning fork test, the monofilament test and the temperature discrimination, the neurological risk status of each diabetic can therefore be determined rapidly and cheaply.

3.7.2 Evaluation of Peripheral Vasculature:

This includes palpation of the pulses. (Dorsalis pedis, posterior tibial; popliteal and femoral) Absence of distal pulses in a diabetic foot is a sure sign of significant arterial disease. However, presence of palpable pulses does not absolutely exclude vascular disease.

Determination of Ankle brachial index using Doppler is a simple method of assessing vascular insufficiency. It is obtained by dividing the ankle systolic pressure by the brachial systolic pressure. Normal values are 1.0 ± 0.1. However, the ABI can be deceptive because calcification of vessels in Diabetes can lead to falsely elevated ABI[28].

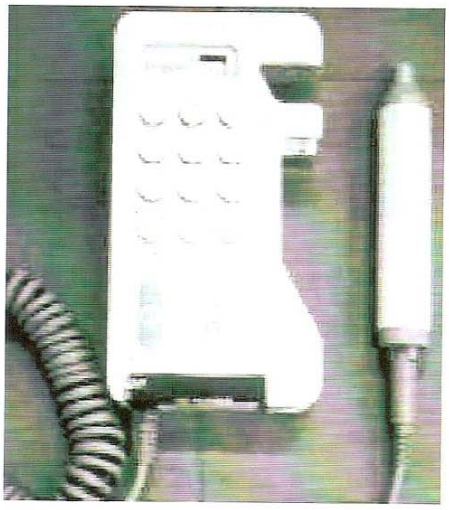
3.7.2.1Duplex Ultrasound

This assesses both anatomical and functional abnormality in the various arterial segments. Significant stenosis is indicated by a peak systolic velocity ratio greater than two across the arterial lesion. Waveform analysis can give additional information about the degree of stenosis.

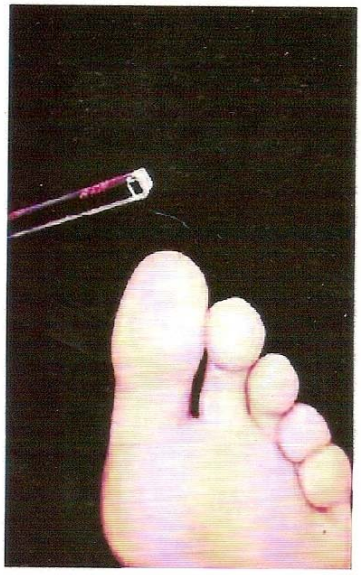
3.7.2.2 Transcutaneous oxygen pressure mapping

This can be used to determine the severity of foot ischemia, thus aiding selection of appropriate treatment. Studies show that if transmetatarsal TcPo2 level is 30 mmHg or greater, treatment should be conservative comprising local wound care, debridement, or a minor ablative procedure. If the transmetatarsal TcPo2 level is below 30 mmHg, it will anticipate the need for vascular reconstruction.

DOPPLER APPARATUS



SEMMES - WEINSTEIN MONOFILAMENT



3.7.2.3Angiography

Angiography remains the `gold standard' for assessment of the lower arterial system prior to any intervention. In can be performed via femoral or brachial catheterisation with iodine - based contrast used to visualise the blood vessels.

3.7.2.4Magnetic resonance angiography

This is likely to be the future investigation of choice. It can detect the occlusion distances. Diabetic patients with poor renal function may benefit from MRA. Studies show that MRA is better than digital subtraction angiography in revealing peripheral run off vessels and patent pedal vessels and suitable for distal by pass grafting.

3.7.2.5Co2 angiography

Also exhibits marked advantages over the procedures currently used through its non - allergising method and the absence of renal toxicity.

3.7.3 Evaluation of Bone Involvement:

Several methods are used for the diagnosis of osteomyelitis.

Plain radiographs have a sensitivity of 55%, but when repeated usually 2 weeks later - the sensitivity is higher, making this the most cost effective diagnostic procedure.

Computerised tomography may reveal areas with subtle abnormalities such as periosteal reactions, small cortex erosions and soft tissue abnormalities.

Magnetic resonance imaging has a sensitivity of almost 100% and a specificity of over 80% and has the potential to reveal abscesses. Therefore this is the preferred method for the diagnosis of osteomyelitis in many centers in cases where the plain radiographs do not provide sufficient information to make a conclusive diagnosis[29]. However, the specificity of MRI decreases in the presence of neuro - osteoarthropathy, prior bone biopsy, recent bone fracture or recent surgery. Magnification radiography is also a very useful method for the detection of early osteomyelitis and it is used to follow up the disease.

^{99}Tc scintigraphy is useful in cases of questionable osteomyelitis. It has a high sensitivity (over 90%) but a low specificity (33%) particularly in the presence of neuro - osteoarthropathy. Although, increased radionuclide uptake during the flow and pool phase is not specific to the diagnosis of osteomyelitis (it may mean soft tissue, bone infection or both), delayed images of the ^{99}Tc scintigraphy showed increased blood flow to the bones only, thus increasing the specificity of the method in the diagnosis of bone infection. Patients with neuro - osteoarthropathy have increased bone blood flow in the absence of osteomyelitis. Like ^{99}Tc scintigraphy, Gallium - 67 citrate accumulates in both osteomyelitis and neuro - osteoarthropathy. This is the reason for its low specificity in the diagnosis of osteomyelitis in diabetic patients. Indium 111 White Blood Cell imaging (^{111}In WBCs) is expensive, time consuming, has poor spatial resolution and does not distinguish soft tissue from bone infection[30].

3.8 MANAGEMENT OF DIABETIC FOOT LESIONS

In 1999, the American Diabetes Association recognised several basic principles of diabetic wound healing.

i. Off - loading

ii. Debridement

iii. Use of appropriate dressings

iv. Medical and surgical treatment of infection

v. Vascular reconstruction and / or amputation or reconstructive foot surgery when necessary.

3.8.1 Off loading or pressure relief devices

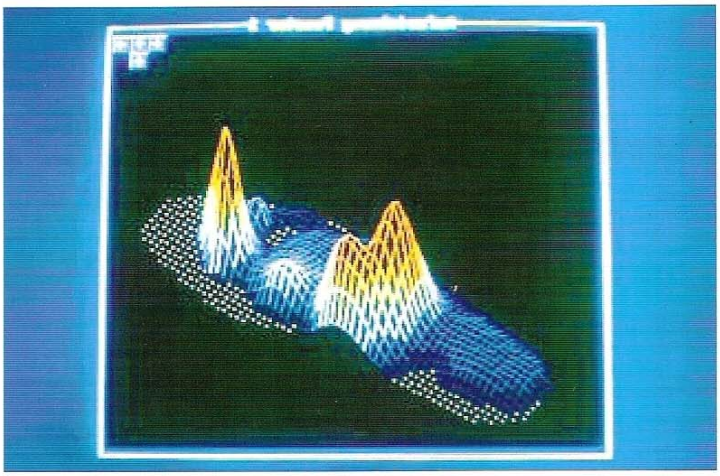
As has already been mentioned, biomechanical changes are a frequent consequence of diabetic neuropathy resulting in an altered pressure load on the sole of the foot. Therefore consistent pressure relief is an essential precondition for the prevention and healing of foot ulcers[31].

Total contact casting (TLC) is the most effective method of off - loading. A total contact cast in a special cast designed to redistribute the patient's weight off the ulcer site allowing ambulation while the ulcer is healing[32].

3.8.1.2Off loading Techniques[33]

Accomodative dressings	-	Patellar tendon - bearing braces
Assistive devices	-	Removable walking braces
Callus removal	-	Scotch cast boot
Foot Casts	-	Shoe cutouts
Half, wedge or surgical shoes	-	Surgical correction of deformity
Orthoses	-	Therapeutic shoes
Padded hosiery	-	Total contact casting

FOOT PRESSURE RECORDINGS



DIABETIC ADAPTED SHOES

3.8.2 Debridement

Debridement of an ulcer is the corner stone of the management of active, acute or chronic wounds. The aim of debridement is to remove fibrin (white, yellow or green tissue seen on the bed of an ulcer) and necrotic tissue (black tissue) and to produce a clean, well vascularised wound bed.

Types of debridement are as follows :-

3.8.2.1 Sharp surgical (using scalpels) the gold standard for wound preparation, removes both necrotic tissue and micro-organisms. Majority of diabetics have neuropathy hence, feel no pain, therefore extensive sharp debridement or even operations on the feet can be performed without anesthesia.

3.8.2.2 Mechanical using wet -to-dry dressings, hydrotherapy, wound irrigation and dextranomers.

3.8.2.3 Enzymatic (using chemical enzymes such as collagenase, papain or trypsin in a cream or ointment base) - chronic wounds are enzymatically debrided in elderly patients when regular, sharp debridement is not possible, e.g. if the necrotic zone is thin, in ulcers with sinuses; and as an additional procedure to sharp debridement.

3.8.2.4 Autolytic debridement; using invivo enzymes which self - digest devitalized tissue such as hydrocolloids, hydrogels, and transparent films. This uses the body's own enzyme and moisture to re-hydrate, soften and finally liquefy hard eschar and slough. It is selective, as only the necrotic tissue is liquified and painless to the patient. Its main indication is non - infected ulcers with mild to moderate exudates.

3.8.2.5Biomechanical wound Treatment : (Biosurgery)

Treatment with sterile maggots (Larval therapy).

The mechanism of action by which the larvae of Lucillia serricata (Greenhottle fly) contribute to the cleaning and healing of necrotically coated or infected wounds has yet to be fully elucidated[34]. The production of an antibiotic - like agent (also effective against micro - organisms resistant to conventional antibiotics), the presence of growth factors in the larval secretion, the destruction of bacteria by absorption and change in the pH value of the wound are postulated.

Leeches (Hirudo medicinalis) are also used in amputation surgery. Because of their local anti - inflammatory and antithrombotic effect due to the formation of hirudin, they are used in areas of critically impaired circulation or in the development of hematoma.

After debridement and infection control, the raw area is allowed to heal by (i) Granulation, (ii) Applying Split skin graft or Local random flaps or Pedicled muscle flaps.

3.8.3 Dressings: Broad spectrum of wound dressing materials currently are available.

LARVAL THERAPY - MAGGOTS DRESSING

Table 2. Advantages and disadvantages of available types of dressings

Type of dressing	Advantages	Disadvantages
Traditional dressings (gauze and absorbent cellulose)	Cheap and widely available. Appropriate for gangrenous lesions	Adhere to the wound bed and may cause bleeding on removal. Provide little protection against bacterial contamination
Films	Semi-permeable. Form bacterial barrier. Durable. Require changing every 4-5 days. Cheap	Useful on flat or superficial wounds only. Some patients are allergic to the adhesive in the dressing
Foams	Appropriate for ulcers with high production of exudates. Provide thermal insulation. Easily conformable, may be used to fill cavities without sinus tracts	Effect difficult to quantify. Not as effective and rapid as surgical debridement. Not appropriate for neuro - ischemic ulcers, which produce minimal exudates. Wound must be monitored closely for signs of infection.
Hydrocolloids	Safe and selective, using the body's own defense mechanisms. Good for necrotic lesions, with light to moderate exudates. May be used to fill cavities wihout sinus tracts. Can be easily used with a shoe. Adhesive surface prevents slippage. Do not require daily dressing changes. Cost – effective	Their occlusive and opaque nature prevents daily observation of the wound. Wound must be monitored closely for signs of infection. May promote anaerobic growth and mask a secondary infection.
Alginates	Useful as absorbents of exudates.Good for infected ulcers. Some products have hemostatic properties.	Not appropriate for neuro-ischemic ulcers,which produce minimal exudates. May dry out and form plug within the wound bed. Requires painstaking removal with the use of large amounts of saline.
Enzymatic Dresssings	Good for any wound with a large amount of necrotic debris, and for eschar formation. Promote autolysis and fast healing. Decrease maceration of the skin and risk of infection.	Costly. Must be applied carefully only to the necrotic tissue. May require a specific secondary dressing. Irritation and discomfort may occur.
Medicated Dressings		Data based on animal models and cell cultures only.

3.9 SURGICAL MANAGEMENT OF DIABETIC FOOT

- Surgical Decompression of foot and leg[35b]
- Role of Amputation
- Role of Vascular Management.

3.9.1 SURGICAL DECOMPRESSION – 3 types.

3.9.1.1FOREFOOT DECOMPRESSION

Webspace infection, central plantar space infection are the indications. Incision should be placed deep into plantar space cutting plantar aponeurosis.

3.9.1.2PLANTAR SPACE DECOMPRESSION

Main indication is a plantar space infection. Characteristic factor of this abscess is disappearance of longitudinal arch and skin crease.The area of longitudinal arch may bulge, sole is edematous. Incision is made from little toe to the heel over the medial aspect.

3.9.1.3FOOT AND LEG DECOMPRESSION(Fasciotomy)

Vertical incision for leg and horizontal for foot abscess, cellulitis are done.

AVERAGE HEALING TIME

Forefoot decompression	=	11 - 38 days
Plantar decompression	=	12 - 40 days
Foot and leg decompression	=	12 - 60 days

3.9.2 ROLE OF AMPUTATION

Factors deciding amputation are[35c]:

1. Age
2. Nephropathy
3. Major vessel disease
4. Gross neuropathy
5. Presence of gangrene
6. Involvement of bone
7. Uncontrolled Diabetic KetoAcidosis
8. Septicemia

3.9.2.1Toe amputation

Patients with demonstrably good circulation are selected for these procedures, indicated when there is gangrene of one digit in the absence of rest pain or when there is a perforating ulcer over the interphalangeal joint of great toe.

3.9.2.2Great toe amputation

Incision made around base of the toe, extended 2 - 3 cms proximally along medial border of the foot. Tendons and tissues are divided and toe dysarticulated through Metatarsophalangeal joint.

3.9.2.3Other toes amputation

Incision at the junction of living and dead tissue. Carefully strip the soft tissues from bone and divide the bone through base of proximal phalanx or dysarticulate at Metatarsophalangeal joint.

3.9.2.4Ray Amputation

Remove the entire toe and the distal half of the metatarsal shaft. Provides excellent drainage of deep parts of foot and removes prominent metatarsal head beneath an ulcer. Indicated for infection involving single Metatarsophalangeal joint arising from trophic ulcer and or infection in deep flexor tendon sheath.

Incision encircles base of toe and extends proximally into the sole. The toe dysarticulated at Metatarsophalangeal joint. The distal part of plantar incision extended down. The metatarsal shaft is shaved off its soft tissue attachment. Bone is divided approximately middle of metatarsal bone. This provides good drainage.

3.9.2.5Trans Metatarsal amputation

For gangrene involving more than one toe and for persistent or recurrent plantar ulcer. Incision is made across the dorsum of foot at the level of middle of metatarsal bone. The plantar incision is at the base of toes. Both are joined along medial and lateral borders.

3.9.2.6 Below knee amputation

Indicated when gangrene and uncontrolled sepsis involves whole foot or lower 1/3 of leg. The posterior flap in lower leg amputation should be long. The bone is divided in the midleg at the junction of upper and middle 1/3 rd of leg. Anterior surface of tibia is bevelled and fibula divided 3 or 4 cms higher than tibia.

3.9.2.7 HIGHER LEVEL AMPUTATIONS

1. Through knee dysarticulation
2. Above knee amputation .

3.10 VASCULAR MANAGEMENT:

3.10.1 Role of pentoxyphylline

A xanthine derivative, decreases blood viscosity and increases red cell flexibility, there by increasing blood flow to microcirculation and enhancing tissue oxygenation.

3.10.2 Antiplatelet Drugs

Platelet aggregation inhibitors like aspirin and dipyridamole decrease progression of atherosclerosis.

Patients who require Emergency Vascular Management.

1. Acute embolic manifestation presenting an cold feet.
2. Acute or chronic occlusion arterial disease presenting with skin discoloration, cellulitis and blebs.
3. Extensive edema necessitates decompression

Revascularisation procedures intervention (dilation) and reconstructive (by pass surgery procedures), should be employed as supplementary measures to preserve the extremity in diabetic foot syndrome.

3.10.3 Endovascular management

Balloon angioplasty with or without stent placement is used for in flow vessel and femoro–popliteral and distal popliteal lesions. The outcome depends

on the site, length, morphology of the lesion and the state of the distal run - off vessels. Currently stents are metallic and permanent and may be self - expanding or require balloon expansion. However, complications may risk losing the entire vessel and associated run - off. At present, there is no clear evidence in the literature to support routine distal balloon angioplasty[36].

3.10.4 Reconstructive procedures

Because of the particular anatomy (the tibial vessels are frequently affected while the ankle and foot arteries are spared), crural (by pass to lower leg vessel) or pedal (by pass to foot arteries) by pass procedures have increasingly obviated the need for amputation in diabetics over the past few years[37].

The favoured graft material is long saphenous vein but arm vein can also be used. In the absence of vein, prosthesis can be used.

3.11 ADJUVANT THERAPY FOR WOUND HEALING

All the below listed therapies are under experimental studies.

3.11.1 Cultured Human Dermis and cultivated equivablents.

Cultured human dermis consists of neonatal dermal fibroblasts cultured in vitro into a bio-absorbable mesh to produce a living, metabolically active tissue containing (N) dermal matrix proteins and cytokines[38]. The effectiveness of these as a treatment for diabetic foot ulcers is also being examined (Dermagraft; Graft skin).

3.11.2 Hyperbaric oxygen therapy

Hyperbaric oxygen (HBO) involves immersing the wound in a pure O2 atmosphere, either with steady or cyclically raised pressure, in a leg chamber or by placing the whole patient in a chamber. A number of possible mechanisms form the rationale for this treatment including improved O2 supply promoting the proliferation of granulation tissue and antibacterial effect on anaerobic organisms[39].

3.11.3 Ketanserin is a 5HT2 serotonergic receptor antagonist reported to inhibit platelet aggregation, block vasoconstriction, improve tissue perfusion and increase granulation tissue formation. It can be administratured orally or topically.

3.11.4 Growth factors Growth factors are applied directly to the wound surface with the intention of stimulating cellular movement, replication and matrix synthesis leading to healing in chronic non - healing wounds, rhPDGF is a recombinant platelet derived growth factor[40]. rbFGF is a recombinant basic fibroblast growth factor using Escherichial colitype B and RGDpm is an ariginine - glycine - aspartic acid peptide matrix[41].

3.11.5 Granulocyte - colony stimulating factor (G-CSF)

G-CSF increases both the production and release of neutrophils from the bone marrow enhancing the ability to fight infection in the blood. While people with diabetes are not neutropenic, diabetes represents an immunocompromised state secondary to neutrophil dysfunction, it is hypothesised that improved neutrophil production and function will improve bactericidal activity in foot ulcers[42].

3.11.6 Electrical Stimulation

Electric current has been shown to facilitate wound healing in animal models and improve blood flow to the foot in vascular studies in diabetic patients.

3.11.7 Sulodexide is an antithrombotic drug being tried for peripheral occlusive arterial disease.

3.11.8 Hyaff : is a semi synthetic ester of hyaluronic acid. Serum or wound exudates when in contact with Hyaff form a moist environment which promotes granulation and healing.

3.12 Tertiary prevention of the diabetic foot syndrome

Patients with diabetic foot syndrome remain at extremely high risk patients for foot complications throughout their life[43]. Health education must include information about what to do in the event of an injury or impending amputation in addition to preventive foot care aspects. Regular after care in special institutions considerably reduces the risk of recurrence of lesions and subsequent amputation in these patients. Numerous studies in the past two years have shown that more than 50% of all amputations in diabetics are avoidable if the following procedures are applied systematically.

- Regular inspection of feet and foot wear of diabetics at each visit to the doctor.
- Preventive foot care and shoe provision in high risk patients and additional education.
- Use of multifactorial and multidisciplinary treatment concepts in the case of foot lesions.

- Early diagnosis and appropriate treatment of peripheral circulatory disorders in diabetics.
- On going after care of patients with previous foot ulcerations or prior amputations.
- Strict adherence to defined indications for amputation and establishment of amputation registers.

3.12.1 General objectives of rehabilitation

1. Increase range of motion, strength, endurance
2. Maximise safety of lower extremity
3. Pain relief
4. Independent ambulation
5. Health education

3.12.2 Temporary measures for ambulation

1. Walker
2. Axillary crutches

3.12.3 Permanent measures for ambulation

1. Below knee prosthesis
2. Above knee prosthesis[44]

3.12.4. Appropriate foot care includes[45]

i. Care of callus, removal of corns, care of interdigital spaces

ii. Treatment of fissures, macerations and fungal diseases

iii. Nail care and the careful correction of nail deformities.

iv. Avoid temperature extremes, test water with elbow before bathing.

v. If the feet feel cold during night wear socks. Do not apply hot water bottes or heating pads.

vi. Do not walk on hot surfaces

vii. Do not walk bare footed

viii. Do not use chemical agents to remove corns and calluses. Do not use strong antiseptic solutions.

ix. Do not use adhesive tape on the feet.

x. Inspect the inside of shoes daily for foreign objects, nail points, linings and rough objects.

xi. Do not soak feet.

xii. For dry feet use a thin coat of lubricating oil.

xiii. Wear properly fitting stockings, change stockings daily.

xiv. Shoes should be comfortable at the time of purchase. They should be made of leather.

xv. Do not wear shoes without socks.

xvi. Do not wear sandals with throng between the toes.

xvii. Cut nails straight across.

xviii. Avoid crossing the legs as this can compress the nerves and vessels.

xix. See the physician regularly to be sure that the feet is examined at each visit.

xx. Notify your physician at once if you develop a blister or a sore.

3.13. IDEAL DIABETIC FOOT TEAM

Diabetic foot requires a team work to save the foot[46]. The team consists of :

1. General Surgeon
2. Dietician
3. Podiatrist
4. General physician
5. Microbiologist
6. Radiologist
7. Pharmacist
8. Nurse
9. Orthopaedician
10. Vascular surgeon
11. Plastic surgeon
12. Physiotherapist
13. Laboratory specialist (foot pressures)

4.0. MATERIALS AND METHODS

4.1 Materials:

This is a prospective study of consecutive Diabetic patients with foot complications admitted in the surgical wards of Government Royapettah Hospital during the period of August 2005 – August 2007. A total of 200 cases were analysed during this period.

4.2 Methods:

Detailed history and thorough clinical examination was done in all cases. Documentation was done using a stratified proforma which included demographic data of the patients studied; all the details of investigations carried out and the types of management and treatments provided to the patients enrolled in the study.

For all patients, hematological, biochemical, microbiological and radiological investigations were carried out as enumerated in the proforma

using standard procedures. Blood sugar and Renal parameters were performed at the time of admission. Fasting, Post prandial, Pre dinner and Post dinner Blood sugar was done on the next day and repeated according to Blood sugar levels. Urine analysis including urine acetone was done . X-ray of local part, Ultrasonogram Abdomen and Hand held Doppler study of both limbs was done. As Duplex scan was not available in our hospital, they were done at Government General Hospital for patients who were suspected to have Arteriopathy.

Appropriate treatment was provided according to the Grade of Diabetic foot lesions. This included infection control with antibiotics only ; Slough excision with antibiotics ; slough excision with split skin graft or flap; fasciotomy; incision and drainage or amputation at appropriate level. All these procedures were carried out as described.[35b,c]

Health Education was given to patients regarding foot care and were followed up regularly every 2 weeks.

Results

& Observations

5.0 RESULTS AND OBSERVATIONS

5.1 Demographic Analysis:

A total of 200 patients with Diabetic foot lesions were admitted and treated during the study period August 2005 to August 2007. The clinical

pattern of foot lesions, Investigations done, treatment given and the associated complications were analysed and the following results obtained.

Table – 1

Age & Sex Distribution

Age	Male	%	Female	%	Total	%
0-19	0	0	0	0	0	0
20-29	1	0.5	0	0	1	0.5
30-39	9	4.5	1	0.5	10	5
40-49	27	13.5	15	7.5	42	21
50-59	37	18.5	13	6.5	50	25
60-69	34	17	27	13.5	61	30.5
70-79	24	12	5	2.5	29	14.5
80-89	6	4	1	0.5	7	3.5
	138	69%	62	31%	200	

Peak Incidence of diabetic foot was seen in the Age group of 50-69years. Increased prevalence was seen among males (69%). In males increased prevalence was seen in the age group of 50-59 years and in females in age group of 60-69 years.

Table – 2

Socio Economic Level of the patients studied.

Socio Economic Class	No. of Patients	%
Poor Socio-Economic Status Monthly Income (< Rs.1000)	110	55%
Lower Middle Class (Monthly Income: Rs. 1000 – 6000)	30	15%
Upper Middle Class (Monthly Income> Rs.6000)	60	30%

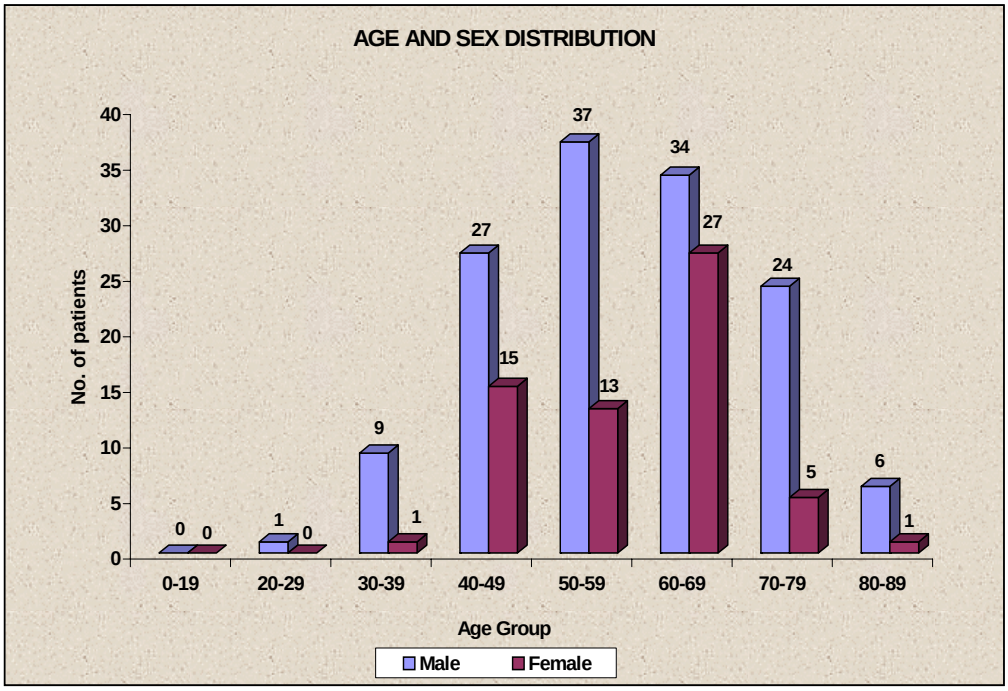
Greater prevalence of diabetic foot was observed in Poor Socio economic class since most of them were manual labourers prone to trauma and due to poor hygiene ($p < 0.001$). Prevalence is also higher in Upper middle class probably due to sedentary life style.

Table – 3

Duration of symptoms before admission

Duration	No. of Patients	%
< week	30	15
7-14 days	50	25
> 15 days	120	60

The above table shows that only less than 15% of patients reported to hospital within 1 week of onset of symptoms. Reasons attributed for delayed reporting for treatment are either that the symptoms were less severely felt by patients due to neuropathy or due to seeking native treatment or due to socio-economic constraints


AGE AND SEX DISTRIBUTION
No. of patients
40
35
30
25
20
15
10
5
0
0 0
1 0
9 1
27 15
37 13
34 27
24 5
6 1
0-19
20-29
30-39
40-49
50-59
60-69
70-79
80-89
Age Group
Male
Female

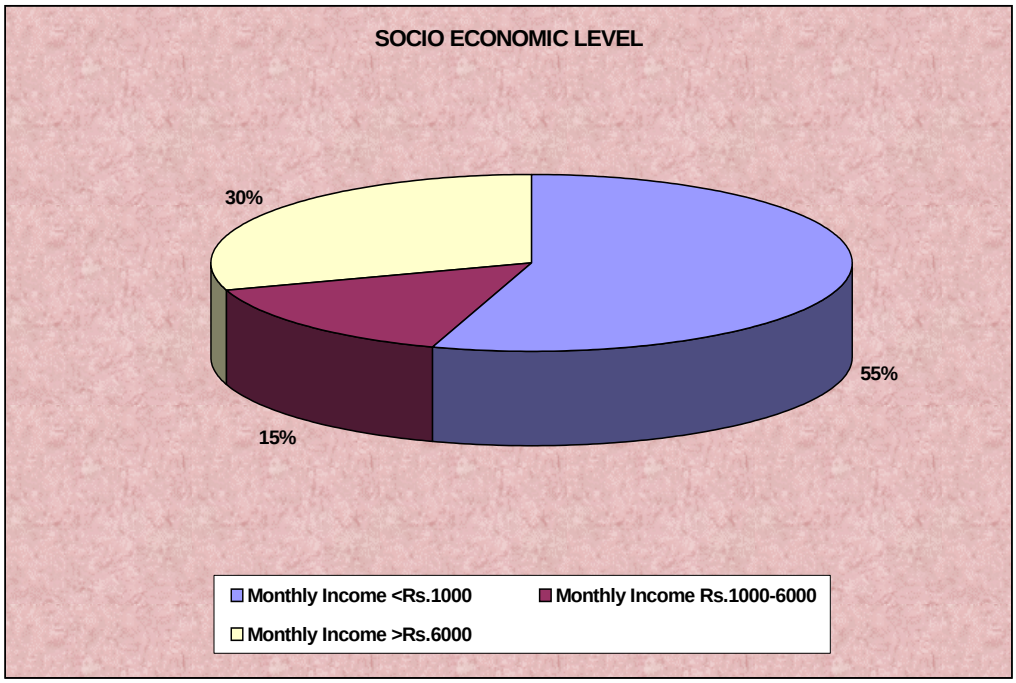

SOCIO ECONOMIC LEVEL
30%
55%
15%
Monthly Income <Rs.1000
Monthly Income Rs.1000-6000
Monthly Income >Rs.6000

Table – 4

Family History of Diabetes

Family History of Diabetes	No. of Patients	%
Present	135	67.5%
Absent	65	32.5%

There was significant family history of diabetes mellites in 67.5% of patients($p < 0.0001$).

Table – 5

Duration of Diabetes before the development of foot lesions .

Duration	No. of Patients	%
Detection at present admission	30	15
< 5 yrs	93	46.5
5-10 yrs	69	34.5
10-15 yrs	7	3.5
> 15 yrs	1	0.5

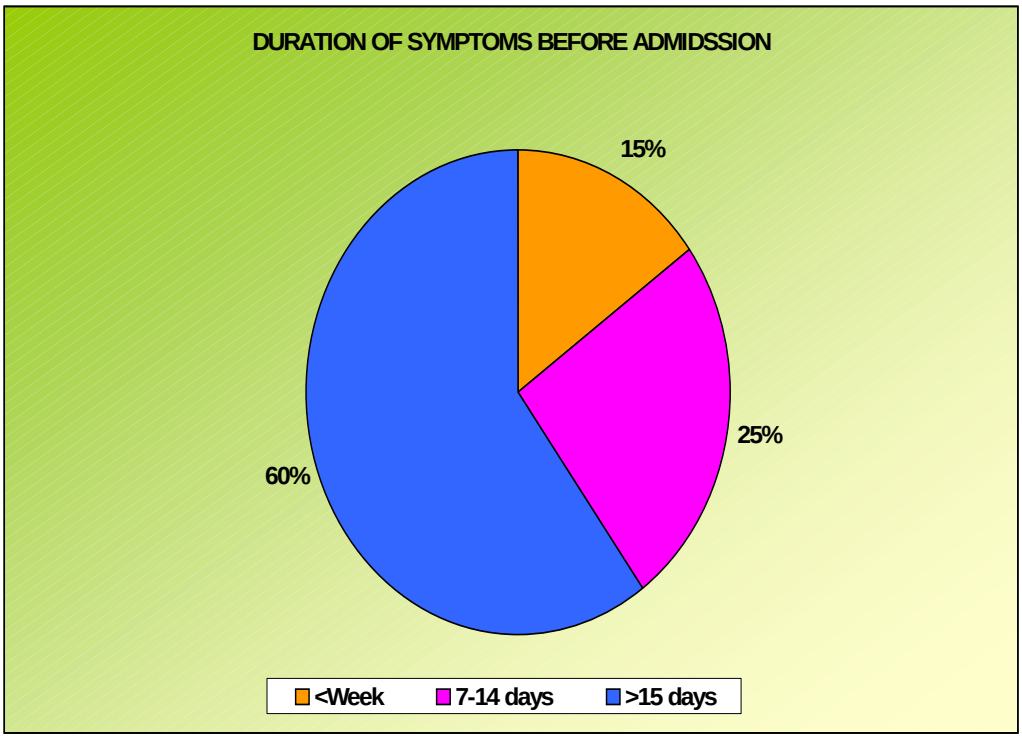
It is observed in this study that 81% of the foot lesions had occurred in patients who have had Diabetes mellitus for more than 1 year to 10 years.

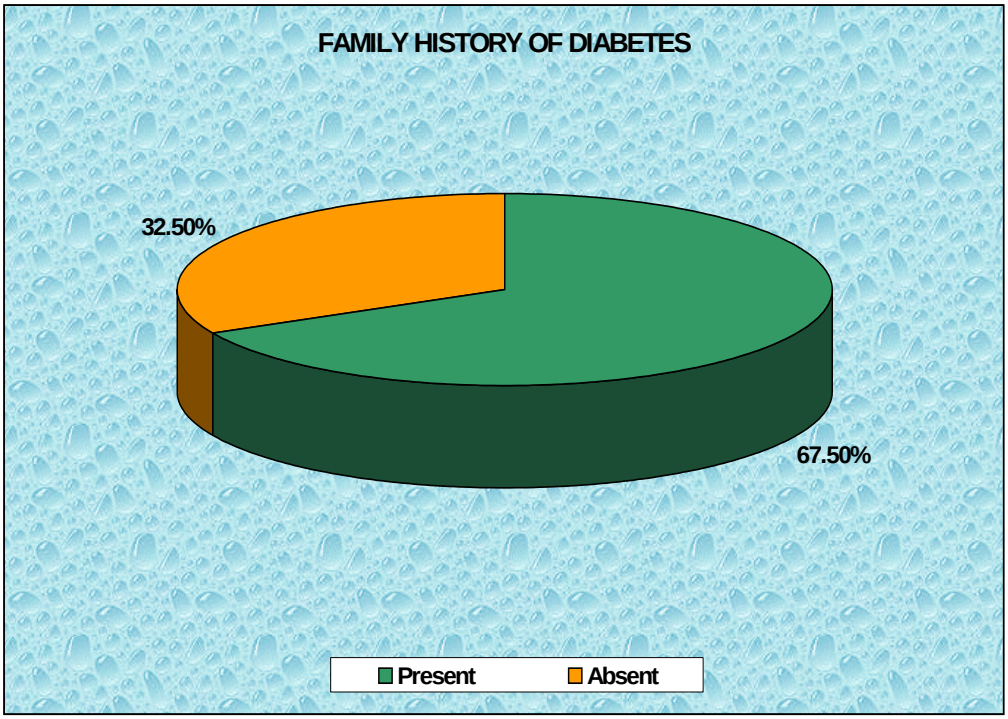
Table –6

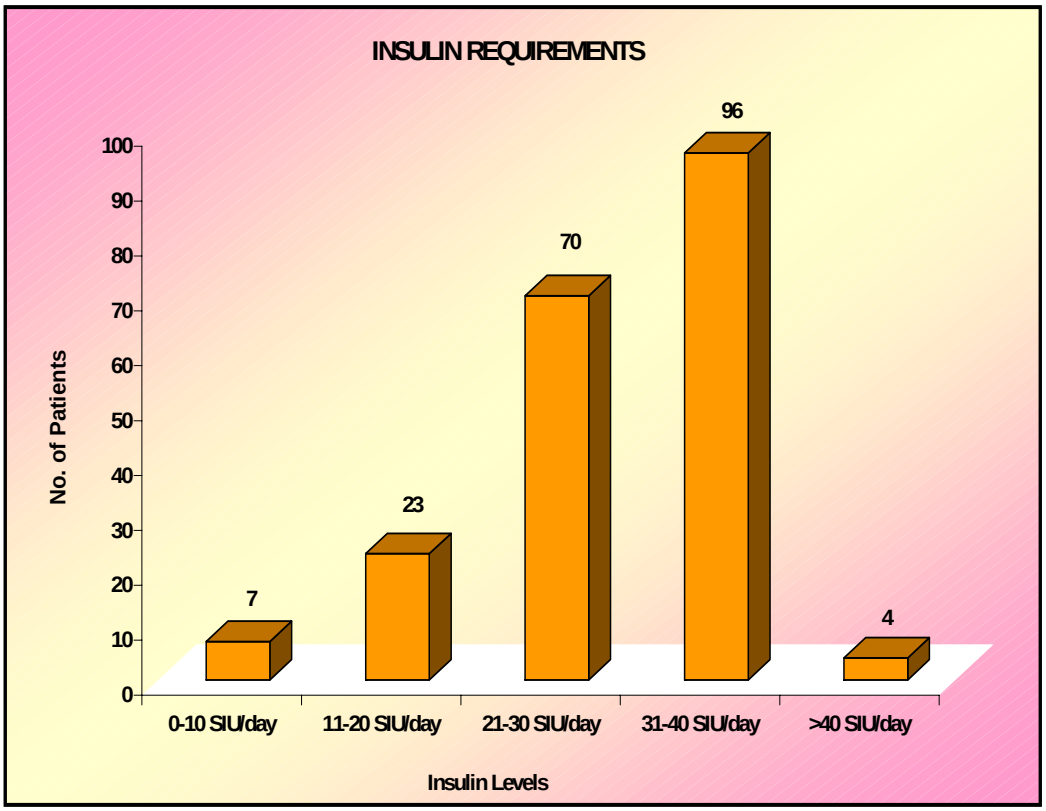
Precipitating Causes

Duration	No. of Patients	%
Spontaneous	60	30
Trauma	140	70

Foot lesions developed either due to trauma or spontaneously. But most of the neuropathic patients would have not noticed or felt the trauma.


DURATION OF SYMPTOMS BEFORE ADMIDSSION
15%
25%
60%
<Week
7-14 days
>15 days


FAMILY HISTORY OF DIABETES
32.50%
67.50%
Present
Absent


INSULIN REQUIREMENTS
No. of Patients
100
90
80
70
60
50
40
30
20
10
0
7
23
70
96
4
0-10 SIU/day
11-20 SIU/day
21-30 SIU/day
31-40 SIU/day
>40 SIU/day
Insulin Levels

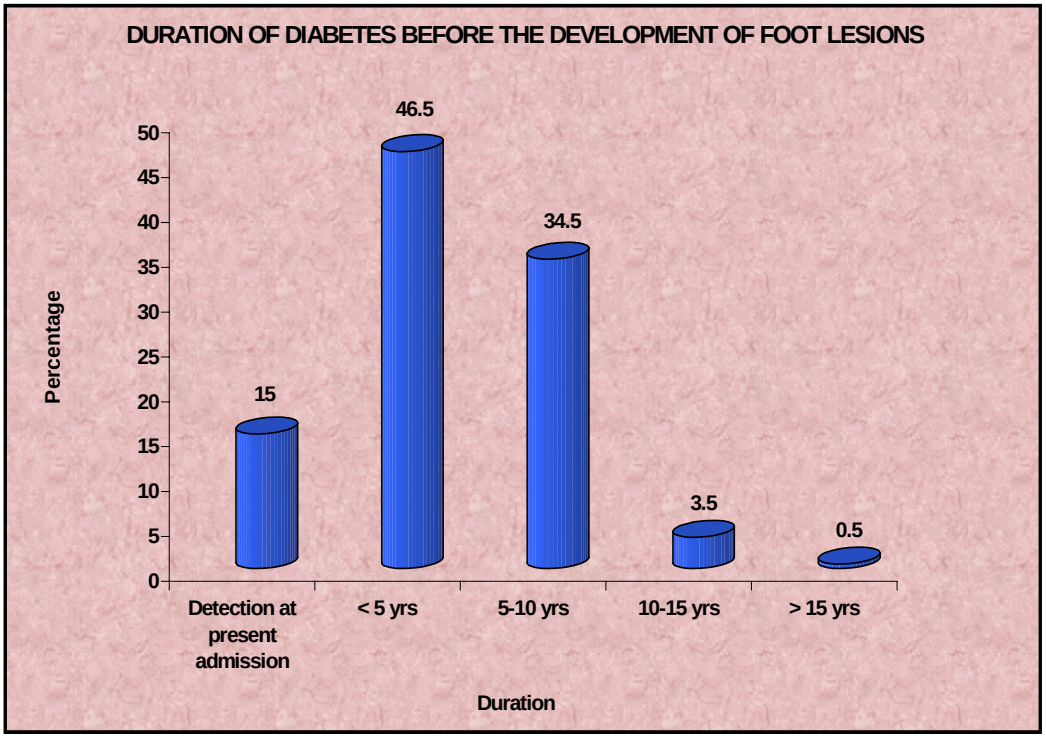

DURATION OF DIABETES BEFORE THE DEVELOPMENT OF FOOT LESIONS
Percentage
50
45
40
35
30
25
20
15
10
5
0
15
46.5
34.5
3.5
0.5
Detection at present admission
< 5 yrs
5-10 yrs
10-15 yrs
> 15 yrs
Duration

Table – 7
Foot wear use among Diabetic foot patients

Percentage	Foot wear
60%	Walk Bare foot
35%	Wear slippers or chappals
5%	Wear shoes

60% of the patients reported to walk bare-footed belonged to poor socio economic class who had increased prevalence of Diabetic foot lesions.

Table – 8

Prevalence of Obesity

Body Mass Index	No. of Patients	%
Normal Range	36	18
High BMI	104	52
Lower BMI	60	30

Most of patients (52)% presenting with Diabetic foot lesions were obese having Body Mass Index higher than normal.

5.2 Profile of Investigations:

Table – 9
Blood Sugar Values on Admission

Random Blood Sugar level (mg/dl)	No. of Patients	%
< 200	50	25
201 – 300	105	52.5
> 301	45	22.5

The analysis showed that about 52.5% of patients had uncontrolled Random Blood Sugar levels on admission in the range of 201 – 300 mg/dl.

Table – 10
Range of Blood Sugar Levels and number of patients in each group.

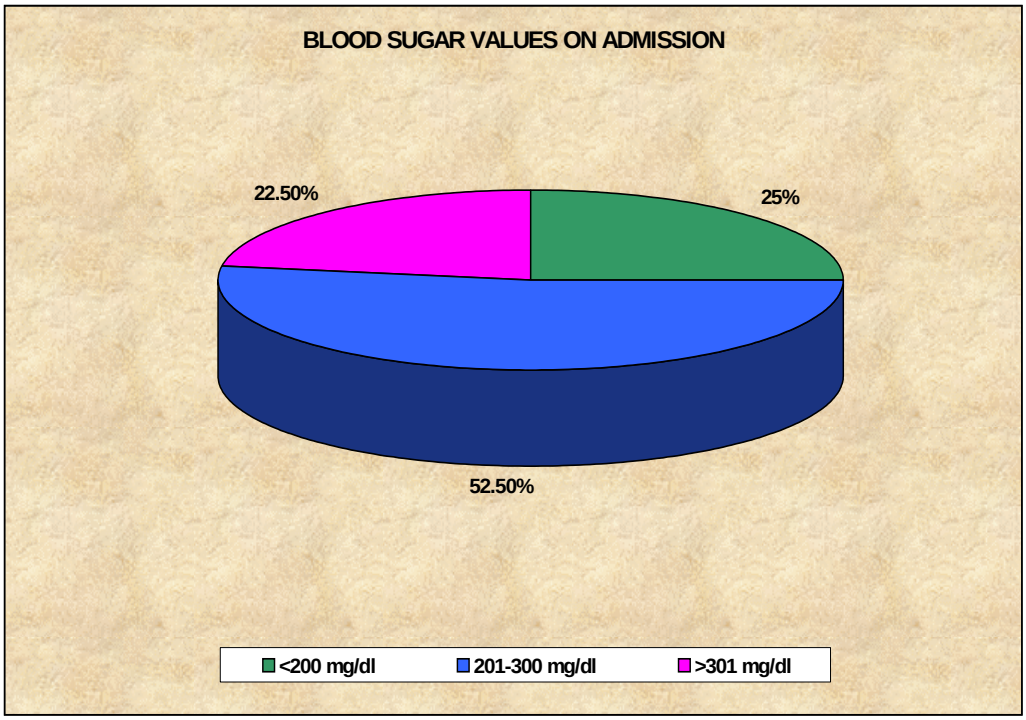
Blood Sugar mg/dl	Random blood sugar	Fasting Blood sugar	Postprandial blood sugar	Pre Dinner blood sugar	Post Dinner blood sugar
< 150	1	1	-	3	-
150-200	49	123	3	110	1
201-250	70	43	43	67	39
251-300	35	27	52	15	57
301-350	42	6	83	4	84
351- 400	3		17		15
> 401	-		2		4

Random Blood Sugar was taken immediately after admission. Fasting, Post-Prandial, Pre dinner and Post dinner blood sugar was carried out on the next day of admission. Insulin dosage was adjusted accordingly to bring about diabetes control . After Glycemic control, Blood sugar was repeated once in three days. It was noticed that the Glycemic status determined the severity of infection. By achieving Glycemic control along with appropriate surgical management, infection was effectively controlled.

Table – 11
Presence of Ketoacidosis in patients presenting with Diabetic foot lesions

Urine Ketones	No. of Patients	%
Positive	43	21.5
Negative	157	78.5

About 21.5% of patients presenting with Diabetic foot lesions had Diabetic Ketoacidosis. Controlling infection was important in treating DKA in these patients.


BLOOD SUGAR VALUES ON ADMISSION
22.50%
25%
52.50%
<200 mg/dl
201-300 mg/dl
>301 mg/dl

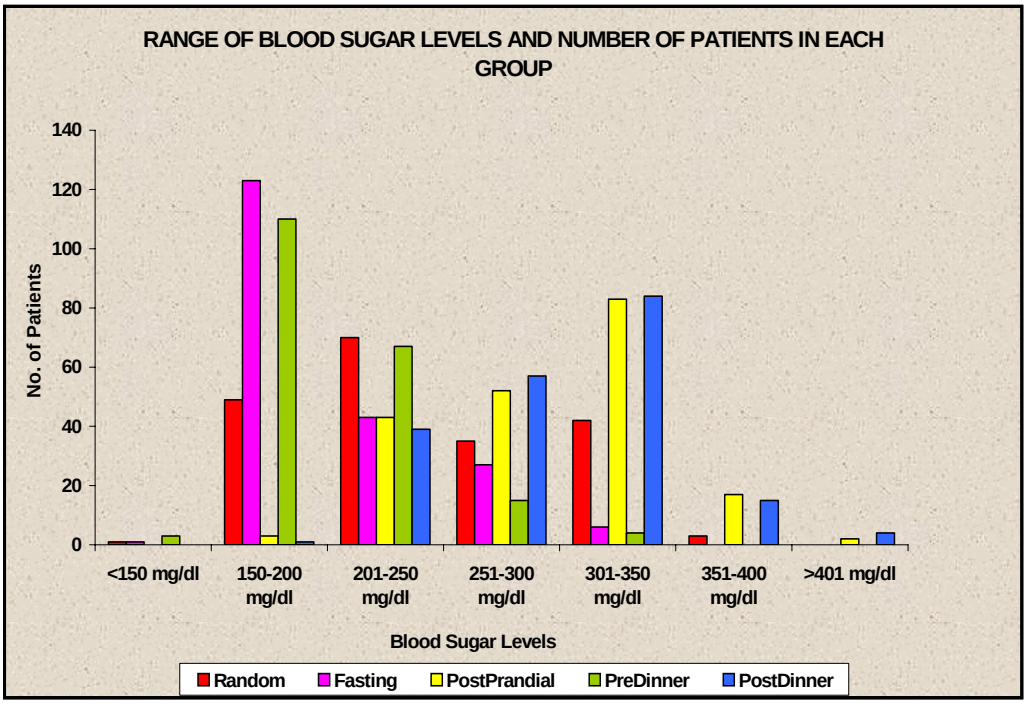

RANGE OF BLOOD SUGAR LEVELS AND NUMBER OF PATIENTS IN EACH GROUP
No. of Patients
140
120
100
80
60
40
20
0
<150 mg/dl
150-200 mg/dl
201-250 mg/dl
251-300 mg/dl
301-350 mg/dl
351-400 mg/dl
>401 mg/dl
Blood Sugar Levels
Random
Fasting
PostPrandial
PreDinner
PostDinner

Table – 12

Prevalence of Anemia Hemoglobin level of 10gm was taken as index

Anemia	No. of Patients	%
Present	122	61%
Absent	78	39%

61% of patients with Diabetic foot lesions had anemia and these patients were given hematinics and Blood transfusion. This enhanced wound healing in them.

Table – 13

Assessment of Renal Parameters

Renal Parameters	No. of Patients	%
Elevated	70	35
Normal	130	65

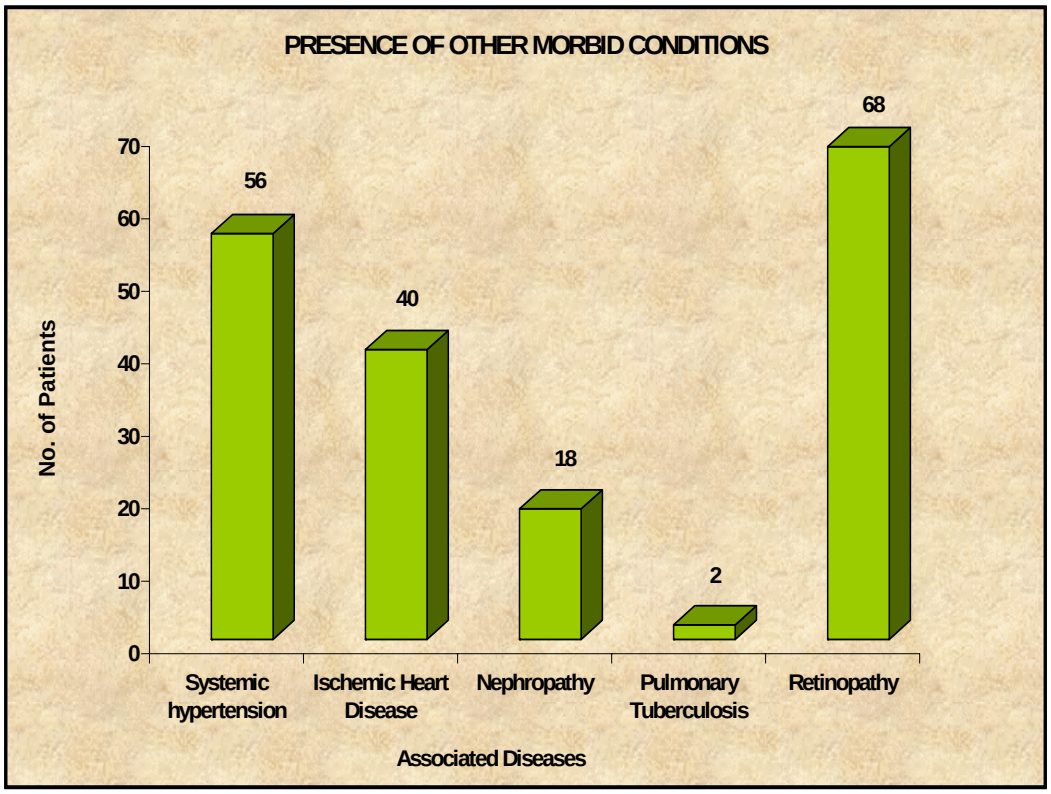
About 35% of patients had elevated Renal parameters. This was either due to dehydration or Associated Diabetic nephropathy.

Table –14a

Assessment of Bacteriology in foot infections

Microorganism Noted	No. of Patients	%
Single Organism	80	73%
Mixed Organism	20	18%
No organizm isolated	10	9%

About 110 patients presented with Diabetic foot ulcers with infection. Pus was sent for culture and sensitivity, of this 73% were of single organisms like Klebsiella, E.coli, Proteus,etc. Only 18% had mixed organism grown in culture.


PRESENCE OF OTHER MORBID CONDITIONS
No. of Patients
70
60
50
40
30
20
10
0
56
40
18
2
68
Systemic hypertension
Ischemic Heart Disease
Nephropathy
Pulmonary Tuberculosis
Retinopathy
Associated Diseases

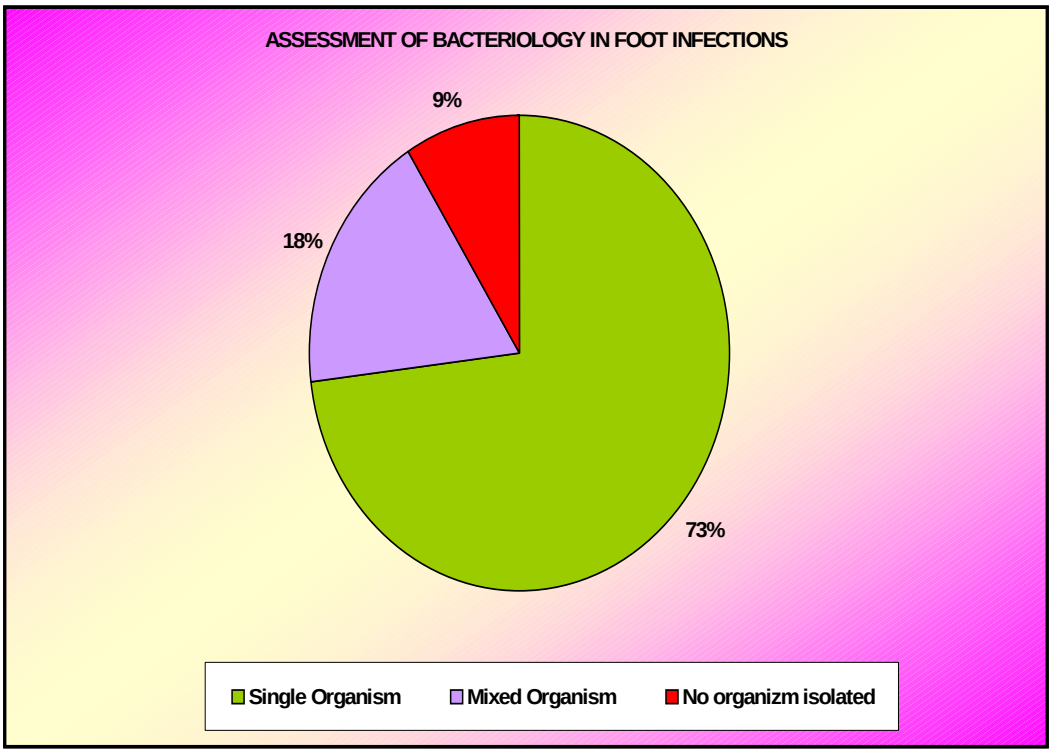

ASSESSMENT OF BACTERIOLOGY IN FOOT INFECTIONS
9%
18%
73%
Single Organism
Mixed Organism
No organizm isolated

Table – 14b
Assessment of Micro organisms present

Micro organism	No. of Patients
Escherichia coli	47
Proteus	48
Klebsiella	16
Pseudomonas	11
Staphylococci	8

Escherichia coli and Proteus were the most common organisms isolated from the Diabetic foot ulcers.

Table 15
Antibiotic Sensitivity

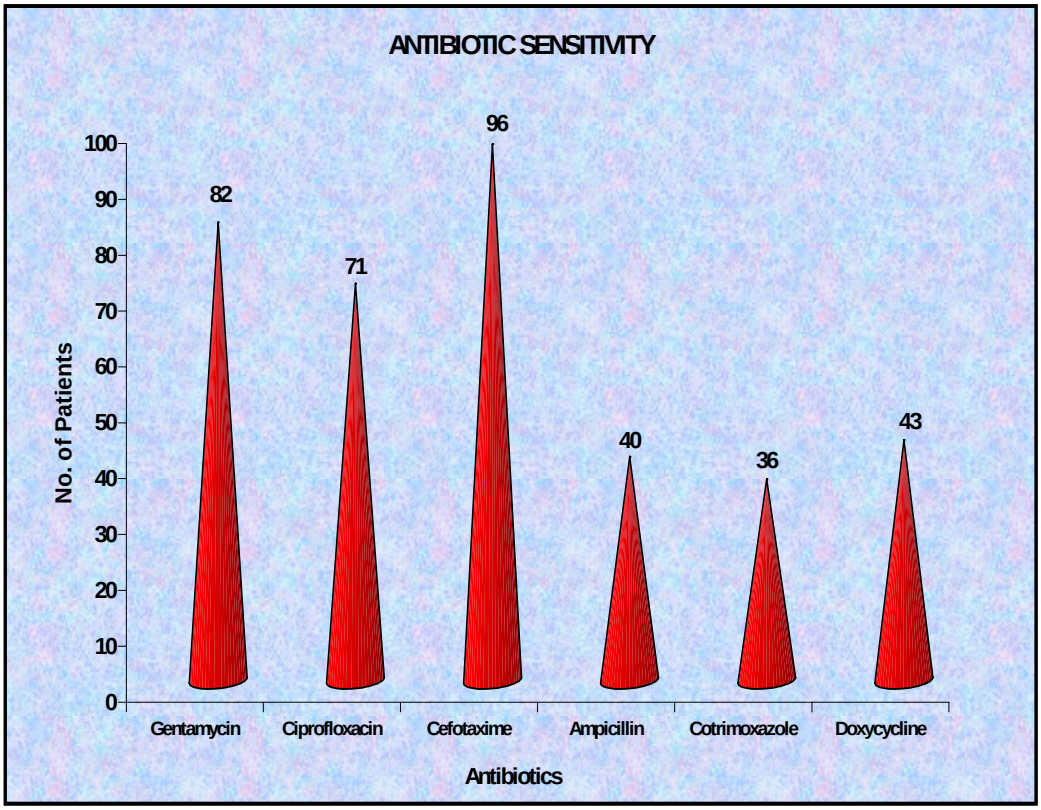
Antibiotics	Sensitive in No. of Patients
Gentamycin	82
Ciprofloxacin	71
Cefotaxime	96
Ampicillin	40
Cotrimoxazole	36
Doxycycline	43

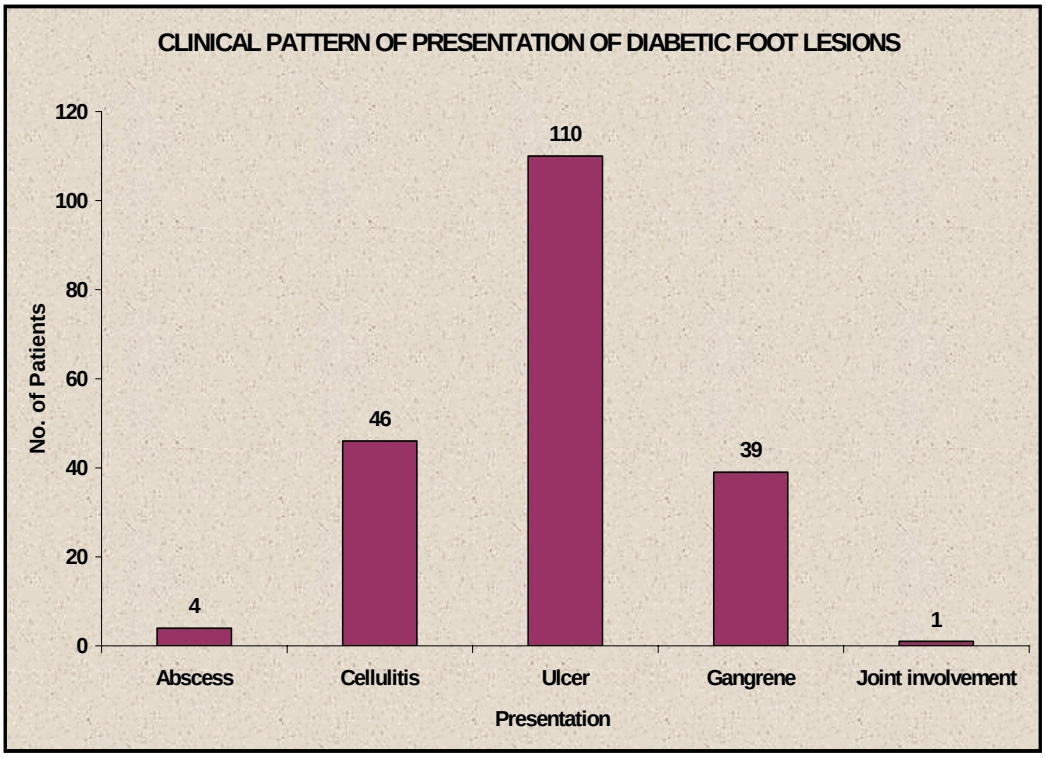
Most patients were found to be sensitive to Ciprofloxacin, Gentamycin and Cefotaxime.

5.3 Clinical Presentation and their Assessment:

Table – 16
Clinical Pattern of Presentation of Diabetic foot lesions

Presentation	No. of Patients	%
Abscess	4	2
Cellulitis	46	23
Ulcer	110	55
Gangrene	39 Toe gangrene 36 Foot gangrene 3	19.5
Joint involvement	1	0.5


ANTIBIOTIC SENSITIVITY
No. of Patients
100
90
80
70
60
50
40
30
20
10
0
82
71
96
40
36
43
Gentamycin
Ciprofloxacin
Cefotaxime
Ampicillin
Cotrimoxazole
Doxycycline
Antibiotics


CLINICAL PATTERN OF PRESENTATION OF DIABETIC FOOT LESIONS
No. of Patients
120
100
80
60
40
20
0
4
46
110
39
1
Abscess
Cellulitis
Ulcer
Gangrene
Joint involvement
Presentation

This shows that about 55% of patients presented with foot ulcers and that toe gangrene was also a common presentation about 36% which if treated earlier can prevent higher level amputation.

Table – 17

Presentation of various Grades of Diabetic lesion (Based on Wagner's Classification)

Grade	No. of Patients
0	-
1	-
2	94
3	20
4	36
5	3

Most of the patients presented with Wagner Grade II type of foot lesions. Hence early and effective management can save the limb.

Table – 18

Clinical Assessment of Arteriopathy

Peripheral Pulses	No. of Cases	%
Absent	16	8
Present	184	92

The above table shows that 8% of patients had macrovascular arteriopathy. These patients were subjected to Duplex Scan. As the facilities for Revascularisation was not available in our Hospital, Six patients were having peripheral arterial disease were referred to Vascular Surgery department, Government General Hospital, Chennai for Angioplasty and Revascularisation procedures for limb salvage.

Table – 19

Prevalence of Neuropathy

Neuropathy	No. of Cases	%
Present	61	30.5
Absent	139	69.5

Neuropathy was present in 30.5% of patients presenting with diabetic foot lesions. Patients with Neuropathy presented with Higher Grades of Diabetic foot lesions.

Table 20

Assessment of retinopathy

Retinopathy	No. of Cases	%
Present	96	48
Absent	104	52

Retinopathy of various grades was present in 96% of patients and this is a risk factor for diabetic foot complications as they are more prone to trauma due to impaired vision.

Table – 21

Assessment of Bone Involvement

No. of Patients subjected to x-ray local part	Bone Involvement	%
200	30	15

15% of patients with diabetic foot lesions had bone involvement either in the form of osteomyelitis, pathological fracture, small joint dislocation or other bony changes.

Table – 22

Prevalence of bony deformities

Bony deformities	No. of Cases	%
Present	78	39
Absent	122	61

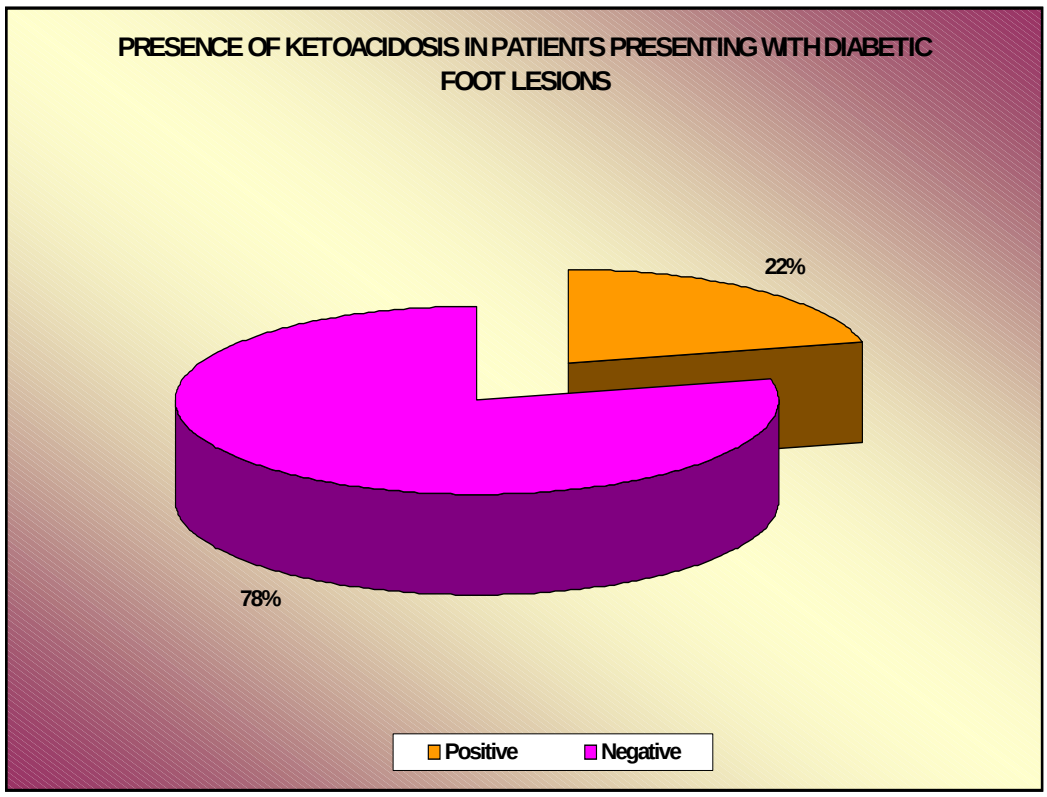
Anatomical Bony deformities was present in 39% of patients which are again an important risk factor for diabetic foot complications.

Table – 23

Prevalence of Risk factors in Diabetic patients.

Risk factor	Present in No. of Cases	%
Family H/o Diabetes	135	67.5
Bony deformities	78	39
Obesity	104	52
Arteriopathy	16	8
Neuropathy	61	30.5
Retinopathy	96	48

Majority of the patients had either one or more of the risk factors leading to complications in Diabetic foot infections.


PRESENCE OF KETOACIDOSIS IN PATIENTS PRESENTING WITH DIABETIC FOOT LESIONS
22%
78%
Positive
Negative

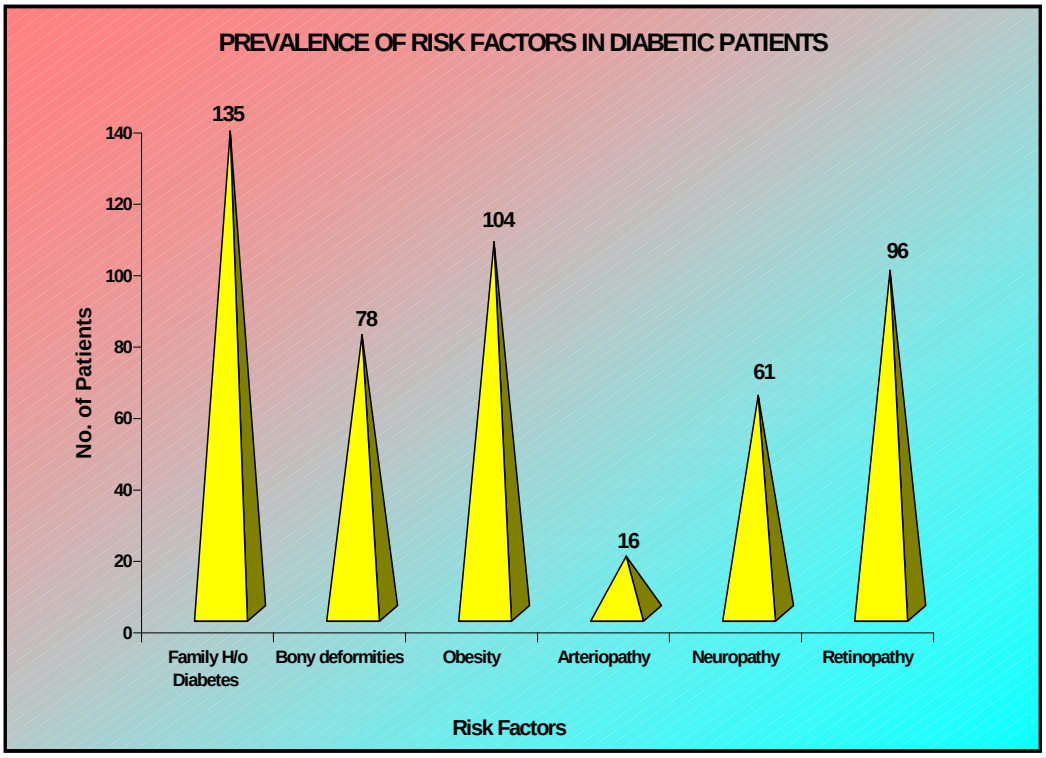

PREVALENCE OF RISK FACTORS IN DIABETIC PATIENTS
No. of Patients
0
20
40
60
80
100
120
140
135
78
104
16
61
96
Family H/o Diabetes
Bony deformities
Obesity
Arteriopathy
Neuropathy
Retinopathy
Risk Factors

Table – 24

Presence of other Morbid Conditions

Associated Diseases	No. of Cases
Systemic hypertension	56
Ischemic Heart Disease	40
Nephropathy	18
Pulmonary Tuberculosis	2
Retinopathy	68

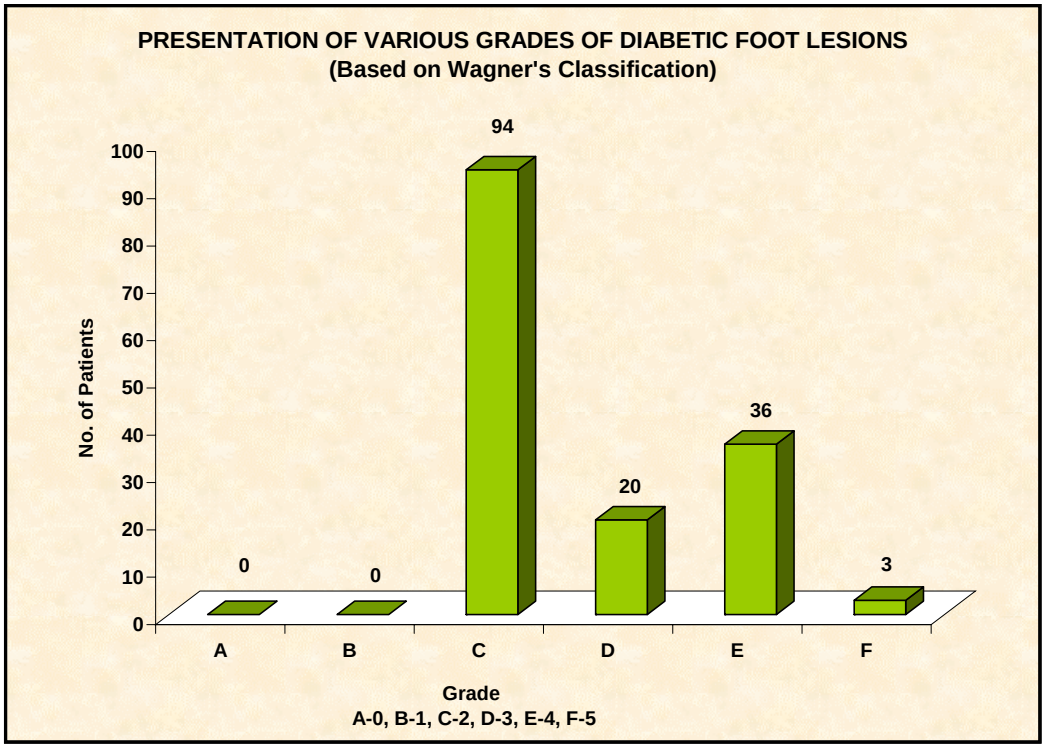
Presence of other systemic complication in patients with Diabetic foot lesions increases the morbidity.

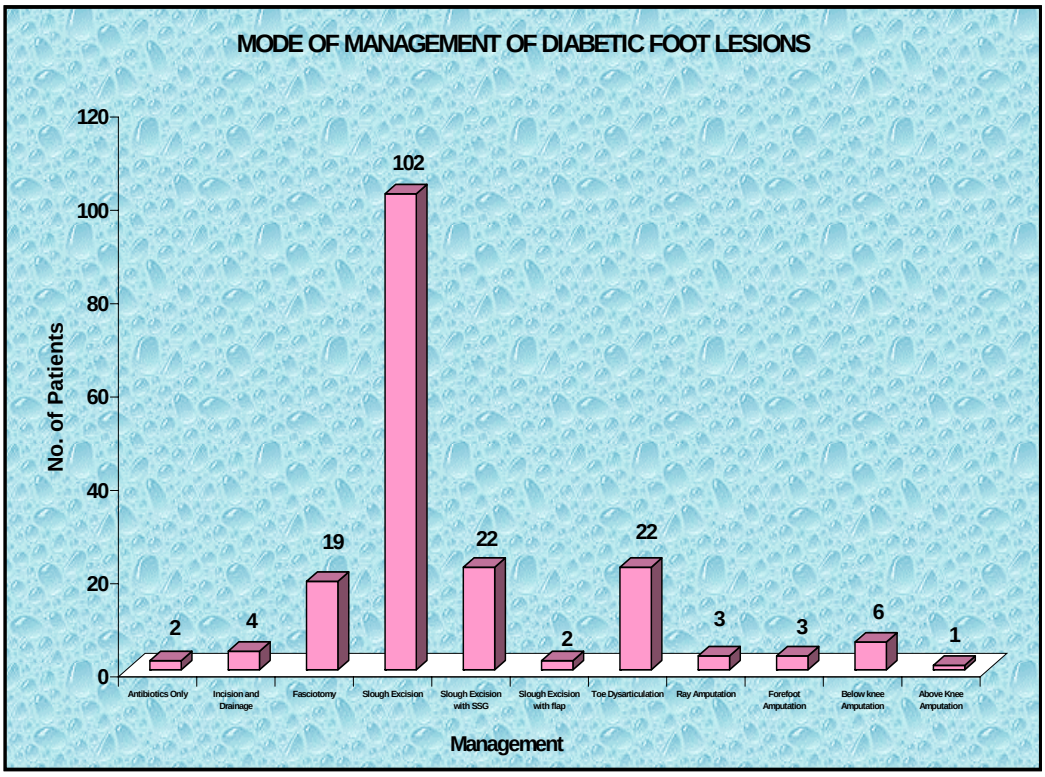
5.4 Management of Diabetic foot lesions of the study:

Table – 25

Management Strategies of Diabetic foot lesions

Management Strategies	No. of cases
Antibiotics Only	24
Incision and Drainage	4
Fasciotomy	19
Slough Excision	102
Slough Excision with SSG	22
Slough Excision with flap	2
Toe Dysarticulation	22
Ray Amputation	3
Forefoot Amputation	3
Below knee Amputation	6
Above Knee Amputation	1


PRESENTATION OF VARIOUS GRADES OF DIABETIC FOOT LESIONS
(Based on Wagner's Classification)
No. of Patients
100
90
80
70
60
50
40
30
20
10
0
0
0
94
20
36
3
A
B
C
D
E
F
Grade
A-0, B-1, C-2, D-3, E-4, F-5


MODE OF MANAGEMENT OF DIABETIC FOOT LESIONS
No. of Patients
120
100
80
60
40
20
0
2
4
19
102
22
2
22
3
3
6
1
Antibiotics Only
Incision and Drainage
Fasciotomy
Slough Excision
Slough Excision with SSG
Slough Excision with flap
Toe Dysarticulation
Ray Amputation
Forefoot Amputation
Below knee Amputation
Above Knee Amputation
Management

Either a single modality or combined modality of treatment was given for effective management. Antibiotics was given to all these patients. Slough excision was done in stages.

Table –26

Insulin Requirements

SIU/day	No. of Cases
0-10	7
11-20	23
21-30	70
31-40	96
> 40	4

Majority of patients required about 21-40 units of Insulin per day. Insulin was given both in plain & monotard form. Dosage was adjusted according to blood sugar levels and thrice daily doses of Insulin was given.

Table – 27

Duration of Hospitalisation

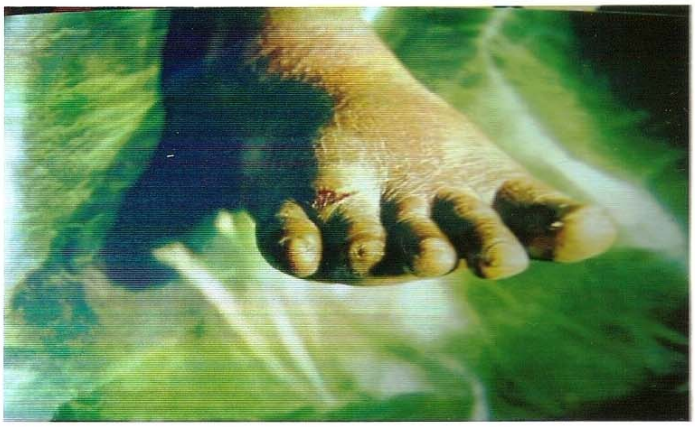
Duration of Stay	No. of Days
Average	27
Maximum Duration	168
Minimum	6

The average number of days of hospitalization was 27. Maximum duration was 168 days complete recovery was seen in 93.5% of patients either after slough excision, disarticulation or Amputation.

13 cases expired or went home against Medical advice or got absconded in the middle of treatment.

Success rate was about 93.5% and mortality rate of 2.5%(5patients) was encountered in our study .Outcome was not traceable in the remaining 4% of patients.

WAGNER GRADE - I DIABETIC FOOT LESION
Patient I.P. No. 830530



WAGNER GRADE - II DIABETIC FOOT LESION
Patient I.P. No. 816030

WAGNER GRADE - II DIABETIC FOOT LESION
Patient I.P. No. 823969

WAGNER GRADE - II DIABETIC FOOT LESION
Patient I.P. No. 820720

WAGNER GRADE - II DIABETIC FOOT LESION
Patient I.P. No. 818484

WAGNER GRADE - III DIABETIC FOOT LESION
Patient I.P. No. 824548

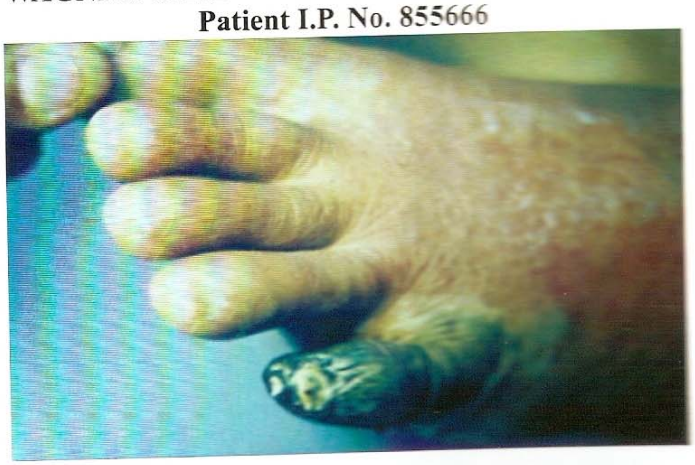
WAGNER GRADE - III DIABETIC FOOT LESION
Patient I.P. No. 853293

WAGNER GRADE - III DIABETIC FOOT LESION
Patient I.P. No. 833461

WAGNER GRADE - III DIABETIC FOOT LESION
Patient I.P. No. 829330

WAGNER GRADE - III DIABETIC FOOT LESION
Patient I.P. No. 822304

WAGNER GRADE - IV DIABETIC FOOT LESION
Patient I.P. No. 855666



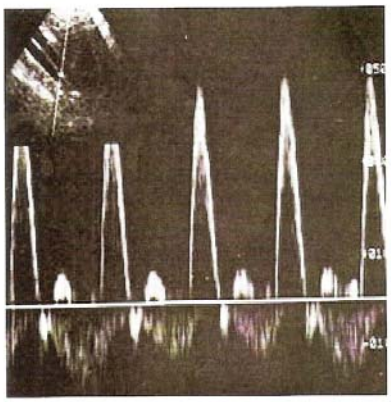
WAGNER GRADE - IV DIABETIC FOOT LESION
Patient I.P. No. 855276

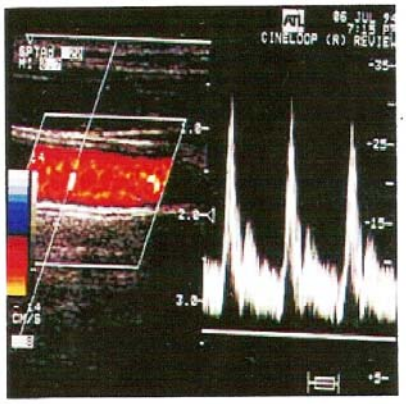
WAGNER GRADE - IV DIABETIC FOOT LESION
Patient I.P. No. 854609

WAGNER GRADE - V DIABETIC FOOT LESION
Patient I.P. No. 874011

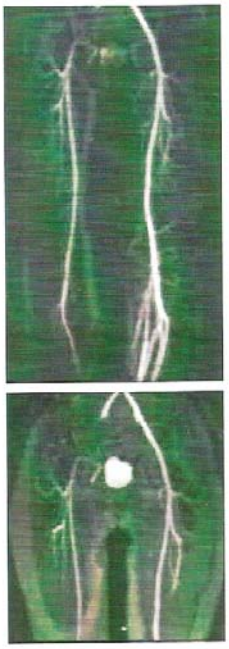
CHARCOT JOINT
Patient I.P. No. 852304

DOPPLER WAVE FORM

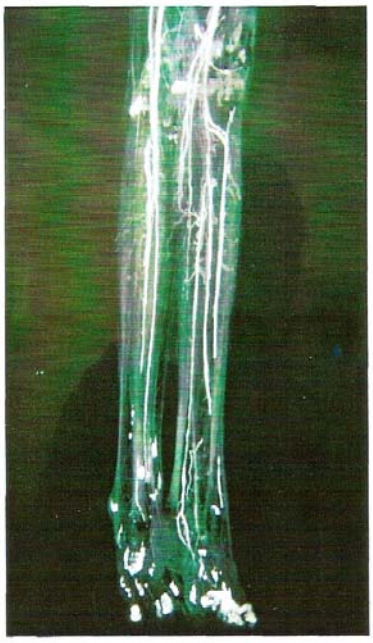




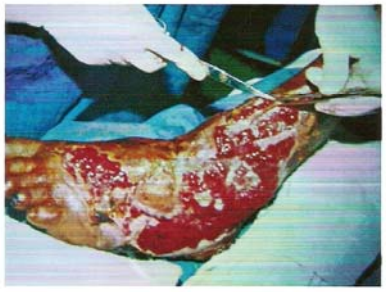
MAGENETIC RESONANCE ANGIOGRAM
SHOWING RIGHT COMMON
ILIAC ARTERY OCCLUSION
Patient I.P. No.861206



CT ANGIOGRAPHY SHOWING OBLITERATED
TIBIAL VESSELS & PATENT PEDAL VESSELS
Patient I.P. No.861100



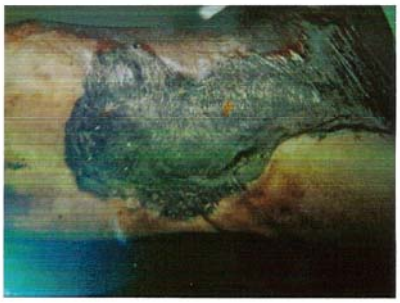
SLOUGH EXCISION BEING DONE
Patient I.P. No.828339



GRANULATED ULCER
Patient I.P. No.876402

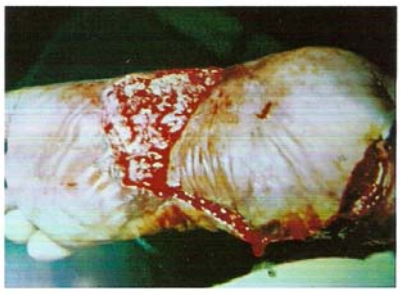
GRANULATED ULCER
Patient I.P. No.877210

SPLIT SKIN GRAFT DONE
Patient I.P. No.837282



FLAP
Patient I.P. No.860311

FLAP
Patient I.P. No.864066



TRANS METATARSAL AMPUTATION
Patient I.P. No.838553

MIDTARSAL AMPUTATION
Patient I.P. No.864849

BELOW KNEE AMPUTATION
Patient I.P. No.863769

CRURAL BYPASS DONE
Patient I.P. No.860712

6.0 DISCUSSION

6.1 Prevalence of Diabetic foot:

The incidence of Diabetes Mellitus is increasing globally. India is emerging as the epicenter of Diabetes today with the current prevalence rate of 14% in the population. Patients with Diabetes have a 12-25% lifetime risk of developing a foot ulcer. Foot ulcers have become a major and increasing public health problem; the morbidities, impairment of the quality of life of patients and the implied costs for management have attracted the attention of health policy providers. Inspite of their rising importance, the management provided for foot ulcers is often inadequate, resulting in delayed healing and eventually the possibilities of amputation. It is projected that developing countries will experience the greatest rise in the prevalence of Type 2 Diabetes in the next twenty years. The people living in these countries, therefore, could expect greater risks of foot ulceration[47].

The present study was conducted in Government Royapettah Hospital in Chennai. In our study of 200 consecutive cases of Diabetic foot, maximum rate of 30.5% was seen in 60-69 years age group, while it was 25% and 21% in the 50-59 and 40-49 years age-groups respectively. The age groups involved in our present study is similar to that reported from Karl Franzens University, Austria(Mean age 66 years) and by Hasbum et al from Mexico Hospital[48] (Mean age 60+/-4 years). A study was undertaken in the USA in 2004 through

the 2002 National Hospital Discharge Survey, looking at 275,000 in patient records from 500 hospitals since 1996. The study revealed that elderly Diabetics had twice the risk of developing a foot ulcer, three times the risk of developing a foot abscess and four times the risk of developing Osteomyelitis[49].

6.2 Causes of diabetic foot:

In our present study, the most common cause of Diabetic foot was trauma in 70% and the remaining as complications of their Diabetic status. In Nigeria, the available hospital studies have variously suggested that trauma or complications of traditional bonesetting and complications from Diabetes Mellitus are the most common causes[50]. It is also observed in our study that 60% of Diabetic foot occurred among those who walked bare foot and 35% in those wearing only slippers or chappals while only 5% prevalence was observed in those wearing shoes. This observation revealed that prone-ness to injuries increased the risk of developing Diabetic foot lesions($P<0.001$)

6.2.1 Diabetic Foot Lesions:

In Wagner's Grade 2 through 5, the overall chance of local or major amputation is estimated to be around 60%. In the present study, the patients with diabetic foot presented with abscess(2%); cellulitis(23%); ulcer(55%) and gangrene(20%). The ulcer pattern ranged from 94% in Grade 2, 20% in Grade 3, 36% in Grade 4 and 3% in Grade 5 category. In earlier studies, Treece et el from City Hospital,[50] UK in their study of 389 diabetic ulcer patients, 78.4% were of Grade 2 type, 10.8% had Grade 3 type and rest Grade 4. Austria reported 22.7% of cases with Grade 2 type and 38.7% with Grade 3 type.

Similarly Hasbum et al from Mexico Hospital[48] have also reported 23% of their diabetic cases with Grade 2 ulcers and 21% with Grade 3. Our study observations are similar to those of Treece et al and Hasbum et al.

6.2.2 Infections:

Infected chronic ulcers may be classified as mild, moderate and severe. Appropriate tissue and bone cultures are useful to guide the use of antibiotic therapy. Gram positive organisms account for the majority of infections, while the prevalence of Methicillin resistant Staphylococcus aureus has become prevalent in recent years. Unachukukwu et al have stated that although gram positive organisms are overwhelming in chronic diabetic ulcers, the polymicrobial nature of bacterial growth should not be ignored in the management planning, especially in developing countries[51].

The pattern of infection as observed in the present study reveals that while 73% of cases were infected with single infection of gram positive organism, 18% of cases had polymicrobial infections. Among these Escherichia coli and Proteus were the predominant micro organisms involved.

Chronic ulcers are frequently co-existing with fungal infections of the foot and it has been said that bacterial infection could be predisposed by fungal infection. Lee et al in 2003 from Korea in a study of 13,271 patients with diabetes have shown that 78.4% have fungal infection of the feet. Among these infections, 70.8% are Tinea pedis type[52]. The investigators, therefore, consider fungal infection a risk factor for foot ulcers.

6.3 Management of Diabetic foot:

The general management and treatment of Diabetic foot ulcers is multidisplinary. Foot ulceration is a complication caused by diabetes and is invariably infected .The Diabetic state, therefore, needs to be well controlled

and infection should be effectively treated. Hence Infection control with appropriate antibiotics becomes a priority. Ulcer care and ulcer surgery is to be considered depending upon the clinical situation and the status of diabetic control.

Although a multitude of factors affect the healing of chronic diabetic foot ulcers, daily or more frequent cleaning and dressing are essential requirements. Regular daily bathing in saline or dilute antiseptic solution offers a better chance of cleaning the ulcers, compared with dressing alone. There are changing perspectives in the local management of Diabetic ulcers which include, apart from new dressings, skin substitutes, growth factors and stem cells.

Despite much efforts towards the treatment of Diabetic foot ulcers, the incidence of lower extremity amputation rate remains about the same. Amputation is a costly outcome and should be prevented as far as possible until otherwise unavoidable.

Amputation of the toe(s) with non healing ulcers or gangrene can sometimes be the only solution towards limb salvage. Amputation, sacrificing the whole leg, is a life-saving procedure for large, unhealed ulcers which are usually accompanied by other complications of neuropathy and ischemia. Patients on the whole would prefer to retain the limb and the attending medical team should be supportive of limb salvage, if feasible.

In the hospital settings, the incidence of amputations, whether minor or major, tends to be higher because of the need for Hospital admission when the

ulcer reaches a more advanced state. The statistics from one General Hospital in Hong Kong indicated that in a ten year period from 1995 to 2005, 154 of the 851 patients admitted with diabetic foot ulcers underwent major lower limb amputations(18.1). In the present study of 200 patients, toe dysarticulation was needed in 22(11%). The types of amputation resorted to were Ray amputation Forefoot amputation in 3 cases each, below knee amputation in 6 and Above knee amputation in 1 case. Majority of the cases could be managed by limb salvage through debridement program of slough excision(102 cases) and slough excision with SSG (in 22 cases), slough excision with flap was resorted in two cases and fasciotomy in 19 cases. Compared to our experience, Hasbeem et al from Mexico Hospital reported amputation performed in 45% of their 377 patients series. Similarly Abhas et al from Tanzania[53] has resorted to amputations in 45% of their 288 cases managed in the Muhimbili National Hospital. However a lower rate of 2.4% amputation was reported by Jeffcoate et al from UK, City Hospital among the 370 patients treated by them which is similar to our observations. Similar amputation rate of 4.9% was also reported by Treece et al from UK, City Hospital[50] in another series of 389 patients.

6.4 Limb Salvage Programme:

Thus it emerges that limb salvage programme in diabetic ulcers with early debridement might significantly reduce the need for amputations of some extent. However prevention of ulcer formation need to be given priority in Diabetic foot management. Consideration of social and psychological implications are also important in planning strategies for prevention of ulcer recurrences. Since the Diabetic foot ulcer has developed into a public health problem, it deserves a holistic approach including socio-economic planning and rehabilitation.

7.0. SUMMARY AND CONCLUSIONS

7.1 Summary:

200 consecutive Diabetic patients with foot complications admitted in Government Royapettah Hospital, Chennai during August 2005 to August 2007 where characterized for demographic factors, investigatory profiles, clinical presentations as per International norms; and underlying risk factors. Effective management strategies were planned and executed with the sole aim of achieving diabetes control and salvaging the diabetic foot with significant success.

7.2 Conclusions:

7.2.1). 76.5% of the diabetic foot cases were in the 40 – 69 years age groups, while maximum cases in men was seen in 50 – 59 years age group and the same in women was in the 60 – 69 years age group.

7.2.2). 55% of the diabetic foot patients were poor and 60% were walking bare-footed and hence prone for trauma and ulcer development.

7.2.3). 52.5% of the cases had high blood sugar levels of 201 – 300 mg/dl at the time of admission and 21.5% had Keto-acidosis.

7.2.4). 91% of the patients were bacteriologically positive for infection either with single organism(73%) or with multiple organisms(18%).

7.2.5). The patients with diabetic foot presented with abscess(2%); cellulitis(23%); ulcer(55%) and gangrene(20%). While 110/200(55%) cases

presented with diabetic ulcers, 85.4% of these cases presented with Grade II ulcers as per Wagner's Classification.

7.2.6). Anatomical Bony deformities, arteriopathy, neuropathy, retinopathy, obesity were the common risk factors in the study group leading to complications in diabetic foot.

7.2.7). In this study, 165/200(82.5%) could be limb-salvaged with antibiotics alone and/or slough excision in various stages and fasciotomy.

7.2.8). 35 cases(17.5%) have to undergo different levels of amputation within which majority of them(22/35) were only toe Disarticulation.

7.2.9). The present study concludes that adequate glycemic control, appropriate antibiotic therapy and prompt slough excision–mediated debridement therapy can be the successful limb salvage programme in nearly 93.5% of the diabetic foot cases.

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Annexure

CLINICAL PROFORMA

Name : Age : Sex : IP No :

DOA : DOD : Duration of Stay :

Presentation : Ulcer [ ] Ulcer with Infection [ ]

Deep Ulcer [ ] Deep ulcer with tendon involvement [ ]

Bony involvement [ ] Gangrene [ ]

Cellulitis [ ]

Site of foot ulcer : Great toe Ball [ ] Other toe [ ]
Heel [ ] Dorsum [ ]
Metatarsal head [ ] Ankle [ ]
Others [ ]

Duration of ulcer at admission <7 days [ ] 7 - 14 days [ ]
>15 days [ ]

H/o. Injury : Yes [ ] No [ ]

Systemic symptoms on admission : Yes [ ] No [ ]

Diabetic Status

Newly detected

0-5years [ ] 5-10years [ ] 10-15 years [ ] >15years [ ]

Treatment for diabetes : Regular [ ] Irregular [ ]

Treatment : Sulfonyl urea [ ] Biguanides [ ]
Combination [ ] Insulin [ ]
Insulin + OHA [ ]

Smoking History : Yes ( ) No( )

Alcoholism : Yes [ ] No [ ]

Nature of work : Manual [ ] Sedentary [ ]

Other concomitant
Illness : HT [ ] IHD [ ] PT [ ]
Nephropathy [ ] Retinopathy [ ]

Drug History : B-Blocker [ ] Calcium channel blocker[ ]
ACE inhibitor [ ] Others [ ]

Place of living : Urban [ ] SemiUrban [ ] Rural [ ]

Knowledge about diabetic
foot problems Aware [ ] Ignorant [ ]

Source of knowledge : Massmedia [ ] Doctor[ ]
Nurse [ ]
Other health worker [ ]

Nature of foot wear used : Nil [ ] Any type [ ]

H/o. Diabetes in family: Yes [ ] No [ ]

H/o. Diabetic foot problem
in family : Yes [ ] No [ ]

Past history of amputations
for foot problems : Yes [ ] No [ ]

Anthropometry :

Body Wt. in Kgs : Ht : BMI : Wt / Ht^2
Blood pressure:

General examination:

Local examination of the foot lesion: Ulcer size, extent, edge,depth, base.

Evidence of peripheral vascular disease : Yes ( ) No ( )

Dorsalis pedis Artery Palpable : Yes [ ] No [ ]
Impaired [ ]

Posterior tibial Artery palpable : Yes [ ] No [ ]
Impaired [ ]

Popliteal Artery palpable : Yes [ ] No [ ]
Impaired [ ]

Femoral Artery palpable : Yes [ ] No [ ]
Impaired [ ]

Ankle brachial index _____

Evidence neuropathy

a. Proprioception : Yes [ ] No [ ]
Impaired [ ]

b. Vibration sensation : Yes [ ] No [ ]
Impaired [ ]

c. Two point discrimination : Yes [ ] No [ ]
Impaired [ ]

At Admission laboratory profile :

Blood Sugar – Fasting, Post prandial, Predinner and Postdinner
Serum Cholesterol
TGL LDL
HD L VLDL
Hb %

Evidence of azotemia : Yes [ ] No [ ]
Albuminuria : Yes [ ] No [ ]
Urine Sugar : Yes [ ] No [ ]
Urine Acetone : Yes [ ] No [ ]

In infected ulcer - organisms isolated

a._____ b. _____

Sensitive antibiotic on admission

a. _____ b. _____
c. _____ d. _____

Resistance noted with

a. _____ b. _____

c. _____ d. _____

Results of doppler study

Dorsalis pedis _____

Posterior tibial _____

Popliteal _____

Femoral _____

Evidence of microangiopathy - Retinopathy

Yes [ ] No [ ]

Grade of retinopathy

N [ ] GrI [ ] Gr II [ ] GrIII [ ] Gr.IV [ ]

Chest Xray

Electro-cardiogram

Echocardiogram

Treatment details and complications.

Oral Hypoglycemics or Insulin

Daily dose of insulin needed for diabetic control.

<10 Units [ ] 10-12units [ ] 20-30units [ ]

30 - 40 units [ ] >40 units [ ]

Anti Platelet Drug:

Treatment required :

Antibiotics only [ ]

Dressing [ ]

Debridement [ ]

SSG [ ]

Flap [ ]

Toe amputation [ ]

Transmetatarsal [ ]

Bk amputation [ ]

AK amputation

Outcome of Treatment modalities:

KEY TO MASTER CHART

DOS	**:**	Duration of stay
SES	**:**	Socioeconomic status
P	**:**	Poor
L	**:**	Lower Middle Class
M	**:**	Upper Middle Class
BMI	**:**	Body Mass Index
FOD	**:**	Family H/O Diabetes
Dur.DM	**:**	Duration of Diabetes Mellitus
FBS	**:**	Fasting Blood Sugar
BD	**:**	Bony Deformities
N	**:**	Neuropathy
V	**:**	Vasculopathy
B	**:**	Bone Involvement
DKA	**:**	Diabetic KetoAcidosis
R.	:	Retinopathy
A	**:**	Anemia
Micro	**:**	Microbiology
Antibiotic	**:**	Antibiotic Sensitivity
Gr	**:**	Grade(Based on Wagner's Classification)
R	**:**	Right
L	**:**	Left
#	**:**	Fracture
Ps	**:**	Pseudomonas
St	**:**	Staphylococci
K	**:**	Klebsiella
Ec	**:**	Escherichia coli
Pr	**:**	Proteus
Cf	**:**	Cefotaxime
Cp	**:**	Crystalline Penicillin
G	**:**	Garamycin
A	**:**	Ampillicin
D	**:**	Doxycycline
S	**:**	Co-trimoxazole

S.No	Name	Age	DOS	SES	BMI	FOD	Dur.	FBS	BD	N	V	B	DKA	R.	A	Micro	Antibiotic	Lesion	Management
		/Sex					DM												
1	Dhanalaxmi	60 F	46	P	<	+	10	200	+	+	-	-	-	+	+	Ps	G,Cp,Cf	GrII Ulcer L foot	Slough Excision
	815398																		
2	Nijamudeen	70 M	36	L	N	+	5	210	-	+	-	-	-	-	+	k	Cp,Cf	GrII Ulcer R foot	Slough Excision
	815427																		
3	Elumalai	70 M	48	P	<	+	6	290	-	-	+	+	+	+	+			Gangrene L foot	Transmetatarsal
	816123																	toes Gr IV	amputation
4	Saraswathy	55 F	50	P	>	+	New	320	+	+	-	-	-	-	+			Cellulitis R leg	Antibiotics
	816898																		
5	Pushpa	45 F	53	P	>	+	2	180	+	+	-	-	+	+	-			Cellulitis L leg	Antibiotics
	817000																		
6	Srinivasan	57 M	22	L	>	+	New	280	+	-	-	-	-	+	+			Cellulitis L leg	Fasciotomy
	817898																		
7	Kullamma	75 F	39	P	<	+	15	150	+	-	-	-	-	+	+	Ec,Pr	G,Cp,Cf	GrII Ulcer L foot	Slough Excision
	817999																		
8	Indirani	60 F	37	U	>	+	5	120	-	-	-	-	-	+	-	Ec,Pr	G,Cp,Cf	GrII Ulcer R foot	Slough Excision
	818484																		
9	Natarajan	59 M	41	P	N	+	8	250	+	-	-	+	+	+	+			Gangrene II toe	Toe Dysarticulation
	818500																	L Gr IV	
10	Sundaram	50 M	19	L	N	+	2	290	-	-	-	-	+	-	-	St	D,Cf,G,A	Abscess R foot	Slough Excision with
	818848																	Gr III	SSG
11	Sakunthala	40 F	39	U	>	+	New	200	-	-	-	-	-	+	+	Pr	G,Cp,D	GrII Ulcer R foot	Slough Excision
	818900																		
12	Bakiyam	72 F	49	P	>	+	2	180	+	+	-	-	-	-	+	Pr	G,S,Cf	GrII Ulcer L foot	Slough Excision
	819148																		
13	Pattammal	40 F	39	U	>	+	5	320	-	+	-	-	+	+	+	Ec	Cp,D,S,G	GrII Ulcer R foot	Slough Excision
	819340																		
14	Gangadaran	85 M	9	P	>	+	15	280	+	+	+	+	+	+	+			Gangrene R	Emergency BK
	819351																	foot Gr V	Amputation
15	Krishnan	65 M	22	P	>	+	5	180	-	-	-	-	-	+	+	Ps	G,Cp,Cf	GrII Ulcer R foot	Slough Excision
	819336																		
16	Saroja	70 F	32	P	<	+	10	300	+	+	-	-	+	+	+	St	D,Cf,G,A	Abscess R foot	Incision & Drainage
	819292																	Gr III	

17	Sakunthala	70 F	35	P	>	+	8	180	-	-	-	-	-	+	+	Ec	G,D,Cp	GrII Ulcer L foot	Slough Excision
	819756																		
18	Devandran	80 M	34	P	<	-	8	150	+	+	-	+	-	+	+	Ps	Cf,D,Cp,G	GrII Ulcer L foot	Slough Excision
	819801																		
19	Sankariah	60 M	8	P	>	+	6	210	-	+	-	-	-	-	+	K	Cf,Cp	GrIII Ulcer L foot	AMA
	819874																		
20	Thomas	55 M	10	U	>	+	5	280	+	-	-	-	+	-	-			Gangrene II toe	R Second Toe
	820628																	R Gr IV	Dysarticulation
21	Srinivasan	55 M	12	U	>	+	New	310	-	-	-	-	-	-	+	Ec,Pr	Cf,Cp,G	GrII Ulcer R foot	Slough Excision
	820720																		
22	Raniammal	61 F	19	P	>	-	6	200	-	-	-	-	-	+	+			Cellulitis L leg	Antibiotics
	820687																		
23	Murugan	29 M	15	U	>	+	4	180	-	+	-	-	-	-	-			Cellulitis R leg	Antibiotics
	820969																		
24	Perumal	70 M	59	P	<	-	10	210	+	+	-	-	+	+	-	Pr	Cf,G	GrII Ulcer R	Slough Excision
	816030																	forefoot	
25	Alamelu	64 M	17	U	>	+	8	200	+	-	-	-	-	+	-	Ec	Cp,D,S,G	GrIII Ulcer R	Slough Excision
	821557																	foot	
26	Komalavathi	60 F	14	P	<	+	5	250	+	-	-	+	-	+	-			IV,V Toe	IV,V toe
																		Gangrene R	Dysarticulation
27	Velludai	40 F	11	U	>	+	New	300	-	+	-	-	+	-	+			Cellulitis L foot	Antibiotics
	822352																		
28	Nagappan	45 M	18	U	>	+	New	270	-	-	-	+	-	-	+	K	Cf,Cp,S,G	GrIII Ulcer R	Slough Excision &
	822304																	foot	SSG
29	Saroja	50 F	6	P	<	+	New	320	-	-	-	-	-	-	+	Ec	Cp,D,S,G	GrII Ulcer R foot	Slough Excision
	823551																		
30	Velammal	61 F	15	P	>	-	6	200	-	-	-	-	-	+	-			Cellulitis R leg	Antibiotics
	823552																		
31	Ponurangan	67 M	8	P	>	+	5	180	-	+	-	-	-	+	+	Pr	Cf,G	GrII Ulcer L foot	Slough Excision
	823969																		
32	Vinayagam	70 M	14	P	<	-	10	180	-	+	-	-	-	+	-	Ec,Pr	Cp,D,S,G	GrII Ulcer R foot	Slough Excision
	824100																		
33	Duraikannu	45 M	15	U	>	+	New	300	+	-	-	+	+	-	-			R IV toe	IV toe Dysarticulation
	824548																	Gangrene	
34	Indrani	60 F	Abs	P	>	+	8	210	-	-	+	+	-	+	-	St	D,Cf,G,A	GrIII Ulcer L foot	Slough Excision

	824548																		
35	Ramar	57 M	49	P	<	+	5	180	-	+	-	-	+	-	+	K	Cf,Cp	GrII Ulcer R foot	Slough Excision &
	824789																		SSG
36	Elizabeth	65 F	49	P	>	+	10	220	+	+	-	-	-	+	+			Gangrene R	R Great toe
	824809																	Great toe	Dysarticulation
37	Chellammal	60 F	7	U	>	-	New	320	-	+	-	-	-	-	+	Ec	G,Cp,S,Cf	GrII Ulcer R foot	Slough Excision
	825486																		
38	Arumugam	58 M	20	U	>	+	7	240	-	-	-	-	+	-	-			Cellulitis L leg	Fasciotomy
	825952																		
39	Raghunath	68 M	15	P	<	-	5	250	+	+	-	+	+	+	-			R III & IV toe	R III & IV toe
	827325																	Gangrene	Dysarticulation
40	Rajammal	50 F	20	U	>	+	New	300	-	-	-	-	-	-	+	Pr	G,Cf	GrII Ulcer L foot	Slough Excision
	828339																		
41	Kailasam	48 M	23	U	>	+	3	210	+	-	-	+	-	-	+			R III toe	R III toe
	828383																	Gangrene	Dysarticulation
42	Saradha	60 F	19	P	<	-	5	200	-	+	-	+	-	+	+	Pr	G,Cf	GrIII Ulcer L	Slough Excision &
	829330																	foot	SSG
43	Ranganathan	70 M	17	P	<	+	8	180	-	+	-	-	-	+	+	Ec,Pr	G,Cp,D,S	GrII Ulcer R foot	Slough Excision
	829180																		
44	Sundaram	70 M	13	P	<	-	5	250	+	+	-	+	+	+	-			L Great toe	L Great toe
	830138																	Gangrene	Dysarticulation
45	Valliammal	55 F	18	P	<	+	New	280	-	-	-	-	-	-	+			Cellulitis R leg	Fasciotomy
	830530																		
46	Murugammal	45 F	16	U	>	+	New	300	-	-	-	-	-	-	+			Cellulitis L leg	Fasciotomy
	830716																		
47	Krishnan	57 M	30	U	>	+	5	200	-	-	-	-	-	-	+			Cellulitis L leg	Antibiotics
	830979																		
48	Mohd.Beevi	75 F	AMA	P	>	-	10	180	-	+	-	-	+	+	+			Cellulitis R leg	Antibiotics
	831032																		
49	Muthulaxmi	60 F	80	P	>	+	8	350	+	+	-	+	+	-	+	St	G,D,Cf,A	GrIII Ulcer R leg	Slough Excision
	832142																		
50	Parasuraman	59 M	32	P	>	+	4	250	-	-	-	-	-	-	+			R II toe	R II toe Dysarticulation
	833306																	Gangrene GrIV	
51	Sugavanam	50 M	13	U	>	+	4	230	-	-	-	-	-	-	+			Cellulitis R leg	Antibiotics
	833727																		

52	Vasantha	40 F	18	U	>	+	5	250	-	+	+	+	+	-	+	Ec	G,Cp,D,S	GrIII Ulcer R leg	Slough Excision
	833946																		
53	Ponamma	72 F	46	P	<	-	15	180	-	-	-	-	-	+	+	k	Cf,Cp	GrII Ulcer L foot	Slough Excision
	832166																		
54	Lakshmi	40 F	18	U	>	+	New	280	-	+	-	-	+	-	+	Ec,Pr	Cp,Cf,G	GrII Ulcer R foot	Slough Excision
	835863																		
55	Munusamy	60 M	13	P	>	+	8	150	-	-	-	-	-	+	-	Pr	Cf,G	GrII Ulcer R foot	Slough Excision
	836165																		
56	Narayanan	70 M	13	P	<	-	10	200	-	-	-	-	-	+	+	Ec	Cf,Cp,G	GrII Ulcer R foot	Slough Excision
	836262																		
57	Veerasamy	75 M	19	P	<	-	10	200	-	-	-	-	-	+	+	Ec	Cf,G,Cp,S	GrII Ulcer L foot	Slough Excision
	836241																		
58	Radha	55 M	12	P	<	+	6	250	-	-	-	-	+	-	-	Pr	Cf,G	GrII Ulcer R foot	Expired
	836236																		
59	Syed Amir	55 M	12	P	>	+	5	180	-	-	-	-	-	-	-	Pr	Cf,G	GrII Ulcer R foot	Slough Excision &
	837282																		SSG
60	Ellamma	48 F	13	U	>	+	3	200	-	-	-	-	-	-	-	Ec	Cf,Cp,S,G	GrII Ulcer R foot	Slough Excision
	837336																		
61	Diilibabu	48 M	40	U	>	+	3	210	+	+	-	+	+	-	+	Ps	Cf,D,Cp,G	GrIII Ulcer L foot	Slough Excision
	833461																		
62	Laxmi	75 F	56	P	<	-	10	150	-	+	-	-	-	+	+	St	G,D,A,Cf	GrII Ulcer R foot	Slough Excision
	837991																		
63	Kutty	75 M	28	P	N	-	10	150	-	+	-	-	-	+	+	Pr	G,A,D,B	GrII Ulcer R foot	Slough Excision
	838020																		
64	Kasim	40 M	23	U	>	+	2	200	-	-	-	-	-	-	-	Ec,Pr	G,D,S,Cf	GrII Ulcer R foot	Slough Excision
	838416																		
65	Radha	50 F	19	U	>	+	6	210	+	-	-	-	-	-	+			R forefoot	R Transmetatarsal
	838553																	Gangrene GrIV	Amputation
66	AbdulKhadar	55 M	19	U	>	+	New	300	-	-	-	-	-	-	+	K	Cf,G,Cp	GrII Ulcer R foot	Slough Excision
	839743																		
67	Annamalai	55 M	20	P	<	+	2	210	-	-	-	-	-	-	+	Pr	Cf,G	GrII Ulcer L foot	Slough Excision
	840306																		
68	Mani	61 M	25	P	<	-	6	180	-	-	-	-	-	+	-	Ec	G,D,S	GrII Ulcer R foot	Slough Excision
	840443																		
69	Veerasamy	70 M	25	P	N	-	10	190	-	+	-	-	+	+	-	Ec	G,Cp,D,S	GrII Ulcer R foot	Slough Excision

	841101																		
70	Shanthi	47 F	14	U	>	+	7	200	+	+	-	-	-	-	-	Pr	Cf,D,G	GrII Ulcer R foot	Slough Excision
	841793																		
71	Ganesan	56 M	28	P	<	+	5	150	-	-	-	-	-	-	+	Ec	G,Cp,Cf	GrII Ulcer R foot	Slough Excision &
	843632																		SSG
72	Vasu	50 M	86	P	>	+	3	150	+	-	+	+	-	+	+	St	D,A,G	GrIII Ulcer L leg	Left Above knee
	844484																		Amputation
73	Saraswathi	60 F	33	P	<	-	5	150	-	-	-	-	-	+	+	Ec,Pr	Cf,G	GrII Ulcer R foot	Slough Excision &
	844849																		SSG
74	Kannan	61 M	8	P	N	0	6	160	-	-	-	-	-	+	+			Cellulitis R leg	Antibiotics
	845281																		
75	Jayaraman	80 M	14	P	<	-	10	180	-	-	-	-	-	+	-	Ec	Cp,D,S,G	GrII Ulcer L foot	Slough Excision
	845950																		
76	Kannan	48 M	17	U	>	+	New	280	-	-	-	-	+	-	+			Cellulitis L leg	Fasciotomy
	846604																		
77	Muskim	52 M	23	U	>	+	New	250	-	+	-	-	-	-	+	Pr	Cf,G,D	GrII Ulcer R foot	Slough Excision
	846572																		
78	Marimuthu	30 M	13	U	>	+	1	200	-	-	-	-	-	-	+	Pr	Cf,G,A,D	GrII Ulcer L foot	Slough Excision
	847112																		
79	Valliammal	50 F	11	U	>	-	5	180	-	-	-	-	-	-	+	Pr	Cf,G,S,D	GrII Ulcer L foot	Slough Excision
	847711																		
80	Mercy	60 F	17	P	>	-	3	150	-	+	-	-	-	+	+	Ec,Pr	Cf,G,Cp	GrII Ulcer L foot	Slough Excision
	868884																		
81	Arumugham	60 M	13	P	<	-	2	180	-	-	-	-	-	+	-			Cellulitis L leg	Antibiotics
	848366																		
82	Ganesan	45 M	9	U	>	+	New	210	-	-	-	-	-	-	-			Cellulitis L leg	Antibiotics
	850991																		
83	Krishnaveni	65 F	10	P	>	-	5	150	-	-	-	-	+	+	-			Cellulitis L leg	Fasciotomy
	852248																		
84	Thillaiamma	60 F	19	P	>	+	4	180	-	+	-	-	-	+	+	Ec	Cp,D,G,Cf	GrII Ulcer R foot	Slough Excision
	849941																		
85	Chinnaponnu	65 F	13	P	N	-	3	200	+	-	-	-	-	+	+	Pr	Cf,G,D,S	GrII Ulcer R foot	Slough Excision
	850446																		
86	Ayyasamy	40 M	31	U	>	+	New	220	-	-	-	-	-	-	+	K	Cf,Cp	GrII Ulcer L foot	Slough Excision &
	847742																		SSG

87	Madhar	44 M	35	U	>	+	2	180	+	-	-	+	-	-	+	Ps	D,Cp,G	GrIII Ulcer L foot	Slough Excision
	848283																		
88	Kuppan	40 M	76	U	>	-	New	250	+	-	+	+	+	-	+			Gr V Ulcer R	R BK Amputation
	849746																	leg	with SSG
89	Mani	47 M	24	P	N	+	5	180	-	-	-	-	-	-	+	Ec,Pr	Cp,G,S,Cf	GrII Ulcer L foot	Slough Excision
	850442																		
90	Sekar	52 M	12	P	<	+	7	200	-	-	-	-	-	-	+			Cellulitis R leg	Fasciotomy
	850460																		
91	Afisuddin	51 M	35	P	>	+	New	250	+	-	-	-	+	-	+			Cellulitis L leg	Fasciotomy
	851086																	with abscess	
92	Kumar	52 M	35	P	<	+	8	200	-	-	-	-	-	-	+	Ps	Cf,D,Cp,G	GrII Ulcer R	Slough Excision &
	851732																	foot	SSG
93	Mani	48 M	18	U	>	+	3	300	+	+	+	-	+	-	+			Charcot joint	L BK Amputation
	852304																	L foot	
94	Kadirvel	59 M	14	P	<	-	6	180	-	-	-	-	-	+	+	Ec	G,D,S,Cf	GrII Ulcer R	Slough Excision
	852417																	foot	
95	Kannan	67 M	14	P	<	-	4	200	-	+	-	-	-	+	-			Cellulitis L leg	Antibiotics
	852922																		
96	Ramkrishnan	56 M	16	U	>	+	2	190	-	-	-	-	-	-	-	Pr	Cf,G,A,D	GrII Ulcer R foot	Slough Excision
	852622																		
97	Vasantha	65 F	11	P	>	-	8	210	+	+	-	-	-	+	+	Pr	Cf,G,A,D	GrII Ulcer L foot	Expired
	853077																	Septicemia	
98	Killiammal	50 F	15	P	>	+	2	240	-	-	-	-	+	-	+	Ec,Pr	Cp,D,S,G	GrII Ulcer L foot	Slough Excision
	852871																		
99	Murugan	45 M	34	U	>	+	New	260	+	-	-	+	+	-	+	Ps	Cf,Cp,D,G	GrIII Ulcer R	Slough Excision
	853293																	foot	
100	Chinnapaiya	60 M	41	P	<	-	10	180	-	-	-	-	-	+	+	k	Cf,Cp	GrII Ulcer R	Slough Excision &
	854090																	foot	SSG
101	Kumar	52 M	29	P	<	+	4	200	+	-	-	-	-	-	+			Gangrene L	L Great toe
	854609																	Great toe	Dysarticulation
102	Meganathan	55 M	17	P	<	+	5	210	+	+	-	-	+	-	+	K	Cf,Cp,S	GrIII Ulcer R	Fasciotomy &
	854688																	foot	SSG
103	Narasimhan	34 M	15	P	<	+	3	180	-	-	-	+	-	-	+			L II toe gangrene	L II toe Dysarticulation
	855276																		
104	Dhandapani	52 M	6	U	>	+	4	150	-	-	-	-	-	-	+			Cellulitis L leg	Antibiotics

	853308																		
105	Krishnan	65 M	14	P	<	-	10	180	-	+	-	-	-	+	+			Cellulitis R leg	Antibiotics
	855346																		
106	Ramasamy	34 M	12	P	<	+	New	200	-	-	-	-	-	-	+			Cellulitis R leg	Fasciotomy
	855697																		
107	Subramani	50 M	14	U	>	+	6	180	-	+	+	-	-	-	-			PVD L LL	Referred to GGH
	855666																		
108	Manickam	30 M	6	U	>	+	New	210	-	-	-	-	-	-	-			Cellulitis L leg	Antibiotics
	855882																		
109	Mani	79 M	35	P	<	-	15	300	+	+	-	+	+	+	-			Gangrene L II	L II toe Dysarticulation
	855923																	toe&ulcer L foot	& Slough Excision
110	Govindan	66 M	11	L	N	-	10	180	-	-	-	-	-	+	-			Cellulitis R leg	Antibiotics
	856306																		
111	Krishnan	60 M	13	P	>	-	8	200	-	-	-	-	-	-	+			Cellulitis R leg	Antibiotics
	855875																		
112	Raju	40 M	15	U	>	+	2	280	+	-	-	+	+	-	+			Abscess R Foot	Incision & Drainage
	857377																		
113	Kannan	46 M	13	U	>	+	5	170	-	-	-	-	-	-	+	Ec	Cp,D,S,Cf	GrII Ulcer R &	Slough Excision
	857968																	L foot	
114	Chinnamani	52 M	12	P	<	+	4	180	+	+	+	-	-	-	+			PVD R LL	Referred to GGH
	858012																		
115	Rani	45 F	12	L	N	+	3	200	-	-	-	-	-	-	+			Cellulitis R leg	Antibiotics
	856149																		
116	Indrani	75 F	12	P	<	-	10	180	-	+	-	-	-	+	+	Ec,Pr	Cp,D,S,Cf	GrII Ulcer L foot	Slough Excision
	865305																		
117	Karpagam	50 F	12	P	<	+	5	190	-	-	-	-	-	-	+	Pr	Cf,G,Cp,D	GrII Ulcer R foot	Slough Excision
	859989																		
118	Kasthuri	43 F	16	U	>	+	3	180	-	-	-	-	+	-	+			Cellulitis L leg	Fasciotomy
	858017																		
119	Mariammal	60 F	32	P	>	-	10	150	-	+	-	-	-	+	+	k	Cf,Cp,D,A	GrII Ulcer R foot	Slough Excision
	859209																		
120	Karpagam	60 F	17	U	>	-	5	150	+	-	-	-	-	+	+	Ec	Cp,S,D,Cf	GrII Ulcer L foot	Slough Excision
	859989																		
121	Ashokkumar	31 M	37	P	<	+	New	240	-	-	-	-	-	-	+			Cellulitis L leg	Fasciotomy & SSG
	858031																		

122	Ponnan	45 M	37	P	<	+	1	200	-	-	-	-	-	-	+	Ps	Cf,Cp,D,G	GrII Ulcer L foot	Slough Excision &
	858492																		SSG
123	Magimairaj	47 M	36	P	<	+	6	220	-	+	-	+	-	-	+	k	Cf,Cp,A,D	GrII Ulcer L foot	L BK Amputation
	858555																		
124	Durai	70 M	29	P	>	-	10	180	-	-	-	-	-	+	+	Pr	Cf,G,A,D	GrII Ulcer L foot	Slough Excision &
	858926																		SSG
125	Balasundar	48 M	7	U	>	+	3	200	+	-	+	-	-	-	+			PVD L LL	Referred to GGH
	859001																		
126	Sivagnanam	50 M	15	P	<	+	4	150	-	+	-	-	-	-	+	Ec	Cp,D,S,Cf	GrII Ulcer R foot	Slough Excision
	860231																		
127	Sampath	51 M	17	P	<	+	5	190	+	-	-	-	-	-	+	Ec,Pr	G,D,S,Cf	GrII Ulcer R	Rotation flap
	860311																	Great toe	
128	Moideen	40 M	6	U	>	+	New	250	-	-	-	-	-	-	+			PVD R LL	Referred to GGH
	860712																		
129	Babu	66 M	6	L	N	-	8	180	-	-	-	-	-	+	+			PVD Both LL	Referred to GGH
	861100																		
130	Anthony	39 M	6	L	N	+	13	180	-	-	-	-	-	-	+			PVD R LL	Referred to GGH
	861206																		
131	Poorani	35 F	17	L	N	+	New	210	+	-	-	-	-	-	-			Cellulitis R leg	Fasciotomy
	861202																		
132	Samuthram	45 F	90	L	N	+	2	180	+	-	-	+	+	-	-			Gangrene R	R Great toe
	865707																	Great toe	Dysarticulation
133	Mohanraj	38 M	23	L	N	+	1	150	+	-	-	-	-	-	-			Cellulitis R leg	Fasciotomy
	861173																		
134	Pazhamalai	55 M	10	L	N	-	5	180	-	-	-	-	-	+	-			Cellulitis L leg	Fasciotomy
	861526																		
135	Arumugham	60 M	55	U	>	-	6	270	+	+	-	+	+	+	+			Intramuscular	Fasciotomy &
	861819																	abscess L leg	SSG
136	Ravikrishnan	72 M	26	P	>	-	10	180	-	-	-	-	-	+	-	Pr	Cf,G,A	GrII Ulcer L foot	Slough Excision
	862307																		
137	Krishnan	48 M	9	L	N	+	2	200	+	-	-	-	-	-	-			Abscess L foot	Incision & Drainage
	862710																		
138	Sheik Nabi	68 M	10	U	>	-	8	250	+	+	-	+	+	+	-			Gangrene R	Ray Amputation R
	863539																	Great toe	Great toe
139	Palani	55 M	25	P	<	+	5	300	+	-	+	-	+	-	-			Wet gangrene	R BK Amputation

	863769																	R lower leg	
140	Rangasamy	80 M	12	P	>	-	12	200	+	-	+	-	-	+	+			Gangrene R	R Midtarsal
	864849																	forefoot	Amputation
141	Rukmani	70 F	11	P	<	-	10	200	+	-	+	-	-	+	-			Gangrene R	Ray Amputation R
	862552																	great & II toe	great & II toe
142	Rahmanbi	63 F	17	U	>	-	8	150	-	-	-	-	-	+	-	Ec,Pr	Cp,G,S,Cf	GrII Ulcer R foot	Slough Excision &
	864066																		Flap
143	Gangadevi	45 M	Abs	L	N	+	2	150	-	-	-	-	-	-	-	k	Cf,Cp,D,S	GrII Ulcer L foot	Slough Excision
	864420																		
144	Sundaramal	65 F	14	P	<	-	8	200	+	+	-	+	+	+	-			R Great toe	R Great toe
	864431																	gangrene	Dysarticulation
145	Babu	65 M	30	P	<	-	5	180	-	-	-	-	-	+	-			Cellulitis R leg	Antibiotics
	865495																		
146	Padma	40 F	Abs	U	>	+	New	210	+	-	-	-	-	-	-	Ec,Pr	G,Cf,Cp	GrII Ulcer L foot	Slough Excision
	866176																		
147	Susai	55 M	7	L	N	-	5	210	+	-	+	-	-	-	-			PVD R LL	Referred to GGH
	866471																		
148	Narasimhan	72 M	33	P	<	-	8	200	-	-	-	-	-	+	-	Ec	S,Cp,Cf,A	GrII Ulcer R foot	Slough Excision &
	866504																		SSG
149	Parmasivam	59 M	168	U	>	-	4	180	+	-	-	-	-	+	-	Ps	D,Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	867675																		
150	Sudailai	60 M	165	P	<	-	5	260	+	+	-	-	-	+	-			Wet Gangrene	R Great toe
	867932																	R Great toe	Dysarticulation
151	Murugesan	60 M	24	U	>	-	5	200	+	-	-	-	-	+	-			Cellulitis R leg	Fasciotomy
	867900																		
152	Murthy	45 M	31	L	N	+	2	220	+	-	-	+	-	-	-			R Great toe	R Great toe
	868108																	Wet Gangrene	Dysarticulation
153	Gangaiyan	50 M	11	U	>	+	4	210	+	-	-	-	+	-	-			Wet Gangrene	Ray Amputation R
	868390																	R Great & II toe	Great & II toe
154	Beer Sahib	80 M	29	P	<	-	20	150	+	+	-	-	-	+	-	Pr	G,Cf,Cp,A	GrII Ulcer L foot	Slough Excision
	874960																		
155	Vedachalam	60 M	AMA	P	>	-	10	240	-	-	-	-	-	-	-	Ec,Pr	S,Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	875117																		
156	Muniappan	55 M	56	U	>	+	5	200	-	-	-	-	-	-	-			Cellulitis R leg	Antibiotics
	875190																		

157	Gunasagar	55 M	22	L	N	-	6	180	-	-	-	-	-	-	+	k	Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	875888																		
158	Annammal	61 F	34	P	<	+	6	180	+	+	-	-	+	+	+			Cellulitis L leg	Antibiotics
	875956																		
159	Mani	30 M	34	L	N	+	1	210	+	-	-	-	-	-	-			Gangrene R	R IV toe
	876210																	IV toe	Dysarticulation
160	Subramani	70 M	28	P	<	-	8	160	-	-	-	-	-	+	-	Ec,Pr	S,Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	876300																		
161	Saroja	64 F	29	U	>	+	9	150	-	-	-	-	-	+	-	Pr	G,Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	876374																		
162	Subramani	85 M	28	L	N	+	15	300	+	+	+	-	+	+	-			Gr V lesion R	R BK Amputation
	874011																	foot	
163	Arumugham	65 M	33	U	>	-	10	200	-	-	-	-	-	+	+	Pr	G,Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	874204																		
164	Radha	70 F	93	P	>	-	8	180	-	-	-	-	-	+	+	St	G,Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	874300																		
165	Raji	65 M	23	P	>	+	5	160	+	-	-	-	-	+	+	Ec	S,Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	874464																		
166	Unnamalai	70 F	28	U	>	-	10	170	-	-	-	-	-	+	+	Pr	Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	874506																		
167	Srinivasan	65 M	9	P	<	-	8	150	-	-	-	-	-	+	+	Ec	S,Cp,Cf,A	GrII Ulcer L foot	Slough Excision &
	874949																		SSG
168	Kasthuri	60 F	24	L	N	+	5	240	+	-	-	-	-	+	+			R Great toe	R Great toe
	868575																	Gangrene	Dysarticulation
169	Usman	63 M	Abs	P	>	-	3	180	-	-	-	-	-	+	+	k	Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	868974																		
170	Parthasarthy	65 M	96	P	>	-	5	160	+	-	-	-	-	+	+	Ps	D,Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	870037																		
171	Attukarar	70 M	95	P	<	-	12	170	-	-	-	-	-	+	+	St	Cp,Cf,A,G	GrII Ulcer R leg	Slough Excision &
	870064																		SSG
172	Srinivasan	59 M	17	L	N	+	8	200	+	+	-	-	-	-	+			Gangrene L	L Middle toe
	870973																	middle toe	Dysarticulation
173	Kumar	60 M	29	P	<	+	10	180	+	-	-	-	-	-	+	Ec	S,Cp,Cf,A	GrII Ulcer R leg	Slough Excision
	871073																		
174	Hagadbasha	60 M	Abs	P	>	+	5	180	-	-	-	-	-	+	-	Ec,Pr	S,Cp,Cf,A	GrII Ulcer R foot	Slough Excision

	871390																		
175	Truman	60 M	6	L	N	+	8	150	+	+	+	-	-	+	-			PVD both LL	Referred to GGH
	871391																		
176	Kasthuri	57 F	18	P	<	+	7	180	-	-	-	-	-	-	-	Ec	S,Cp,Cf,A	GrII Ulcer R foot	Slough Excision
	871078																		
177	Saradha	60 F	70	L	N	+	4	180	+	+	-	-	-	+	-	k	Cp,Cf,A	GrII Ulcer L foot	Slough Excision
	871872																		
178	Thangavel	72 M	6	P	<	+	10	170	-	-	+	-	-	+	+			PVD L LL	Referred to GGH
	872536																		
179	Thambidurai	45 M	30	U	>	+	New	280	+	-	-	-	-	-	+			Cellulitis L leg	Fasciotomy
	872757																		
180	Narasaiah	60 M	63	P	<	+	5	150	-	-	-	-	-	+	+	Ec,Pr	S,Cp,Cf,G	GrII Ulcer L foot	Slough Excision &
	873317																		SSG
181	Umar Sherif	34 M	18	U	>	+	1	190	-	-	-	-	-	-	-	Pr	G,Cp,Cf,A	GrII Ulcer L foot	Slough Excision &
	873658																		SSG
182	Lakshmi	40 F	26	U	>	+	2	210	+	-	-	-	-	-	-			R IV & V toe	R IV & V toe
	876380																	Gangrene	Dysarticulation
183	Muthulaxmi	40 F	29	L	N	+	New	270	+	-	-	-	-	-	-			Cellulitis L foot	Antibiotics
	876396																		
184	Thambu	45 M	39	L	N	+	1	200	-	-	-	-	-	-	+	Ec	Cf,A,G	GrII Ulcer R foot	Slough Excision
	876402																		
185	Basheer	65 M	23	U	>	+	5	190	+	+	-	-	-	+	+			Cellulitis R leg	Antibiotics
	876444																		
186	Saraswathi	50 F	51	L	N	+	4	180	+	-	-	-	-	-	+	Ps	Cf,A,G	GrII Ulcer R foot	Slough Excision
	876900																		
187	Jagadambal	61 F	49	P	>	+	9	180	-	-	-	-	-	+	-	Ec	Cf,A,G	GrII Ulcer R foot	Slough Excision
	876940																		
188	Marumatham	67 M	38	P	>	+	7	160	+	+	-	-	-	+	-			Cellulitis R leg	Fasciotomy
	877204																		
189	Panchatsar	70 M	36	P	>	+	10	180	-	+	-	-	-	+	+	k	Cf,A,G	GrII Ulcer L foot	Slough Excision
	877210																		
190	Rajrathinam	45 M	25	L	N	+	2	200	+	-	-	+	+	-	-			Path # L	L IV toe dysarticulation
	877741																	IV toe	
191	Sornamma	60 F	11	L	N	+	8	210	+	-	-	-	-	+	+	Pr	Cf,A,G	GrII Ulcer L foot	Slough Excision
	877750																		

192	Rajakili	57 M	28	P	>	+	5	270	+	-	-	-	-	-	-			Gangrene R	R great toe
	877900																	Great toe	Dysarticulation
193	Jayalaxmi	65 F	22	L	N	+	6	200	-	-	-	-	-	+	+	Ec,Pr	Cf,A,G	GrII Ulcer R foot	Slough Excision
	878124																		
194	Janaki	60 F	22	L	N	+	5	180	+	-	-	-	-	+	-	Ec	Cf,A,G	GrII Ulcer L foot	Slough Excision
	878124																		
195	Madasamy	58 M	14	P	>	+	8	180	-	-	-	-	-	-	-			Cellulitis L leg	Antibiotics
	878190																		
196	Subbaih	68 M	17	P	>	+	10	230	-	+	-	+	-	-	+			R III & IV Toe	R III & IV toe
	878600																	Gangrene	Dysarticulation
197	Saroja	50 F	25	L	N	+	4	200	-	-	-	-	-	-	-	k	Cf,G	GrII Ulcer L foot	Slough Excision
	878650																		
198	Subramani	48 M	17	P	>	+	2	220	-	+	-	+	-	-	-			R III toe	R III Toe
	878700																	Gangrene	Dysarticulation
199	Kareem	40 M	17	P	N	+	New	280	-	-	-	-	-	-	-	Pr	Cf,G	GrII Ulcer L foot	Slough Excision
	878772																		& SSG
200	Srinivasan	50 M	15	P	>	+	2	180	-	-	-	-	-	-	-	Ec	Cf,G	GrII Ulcer R foot	Slough Excision
	878780																		& SSG